PNEUMONO-DYNAMICS.

BY

G. M. GARLAND, M. D.

ASSISTANT IN PHYSIOLOGY, MEDICAL DEPARTMENT, HARVARD UNIVERSITY.

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To

G. W. GARLAND, M. D.

MY FATHER,

AS A TOKEN OF AFFECTION,

I DEDICATE

This Little Volume.

CONTENTS.

INTRODUCTION

CERTAIN expressions which I have employed in this book have not seemed perfectly clear to some of my friends, and therefore I will define those expressions according to the meaning which I wish them to convey.

HYDROSTATIC LEVEL. — This expression, as is well known, signifies the level which a small body of water assumes when it is contained in an open vessel and in a vacuum chamber. Practically, the level is the same when the water and vessel are exposed to the atmospheric pressure, and therefore the term is applied to all water which is at rest in an open vessel.

HYDROSTATIC EQUILIBRIUM. — Suppose we have a small body of water at rest in an open vessel. If we then lower into that water a plane which is perpendicular to the horizon, but which does not reach to the bottom of the vessel, we shall divide the water into two parts communicating at their base. The various layers of these two parts will stand respectively at the same level, and will mutually balance each other. This condition I call *hydrostatic equilibrium*, and its maintenance depends upon the absence of external disturbing forces, and upon the principles involved in the so-called hydrostatic paradox.

HYDROSTATIC INEQUILIBRIUM. — Suppose the upper surface of our body of water has, by reason of the interference of external forces, assumed an inclination to the

horizontal plane. If we then imagine a plane, which is perpendicular to the horizon, to be passed into the water in the manner above described, it is evident that the two parts of the fluid thus divided will not balance each other, as in the previous case. This condition I call *hydrostatic inequilibrium*. This expression, however, implies nothing regarding the mutual equilibrium which obtains between the water and the external forces operating upon it. They may or may not be in a state of equilibrium, but that point is entirely independent of the one just described.

These terms — hydrostatic equilibrium, and hydrostatic inequilibrium — are, in the sense in which I employ them, almost synonymous with the terms *stable*, and *unstable*, *equilibrium*. To my mind, however, the former terms, as applied to my models, define the idea which I wish to convey more concisely than the latter, and therefore I submit them to the judgment of others.

Negative Pressure. — As I understand it, negative pressure is simply the scientific expression for the common term *suction*. It does not, however, imply anything as to the magnitude or intensity of any force, but simply designates the manner in which that force is applied. Suppose the planes A and B are placed in immediate contact with each other to the exclusion of all intermediate air. Now, if an external force, or if an inherent, automatic force, act upon the plane A to remove it from B, then A is said to exert a negative pressure upon B. The mechanism of the application of negative pressure is very simple.

If the plane A be moved to the left, away from B, by a force x, it is evident that the transmitted atmospheric pressure upon the left side of B will become less than the direct atmospheric pressure upon the right side of the same by the amount x, and therefore B will be impelled

toward A by the amount x. A combined movement of both planes toward the left will accordingly result, provided the resistance of A and B is less than the force exerted.

On the other hand, if a force x be applied to A in such a manner as to push that plane toward B, then the latter will in turn be pushed along, and this is direct or *positive* pressure, and is, under some circumstances, familiarly designated as compression.

The experiments described in this book were performed in the laboratory of Prof. H. P. Bowditch, to whom I owe grateful acknowledgments for many hours of kind assistance, and for many important suggestions. I will add that we always employed some form of anæsthetic whenever we operated upon living animals. I wish, also, to express my obligations to Prof. Calvin Ellis, from whose instruction I derived the first impulse to this study, and from whom I received much valuable advice in the preparation of the clinical features of the book. I must also acknowledge my indebtedness to Mr. F. W. Very, of the Institute of Technology, for his revision of the chapter upon physics.

G. M. GARLAND.

98 Boylston Street, Boston,
December 10, 1877.

PNEUMONO-DYNAMICS.

CHAPTER I.

THE CURVED LINE OF FLATNESS WITH PLEURAL EFFUSIONS.

Percuss a healthy, normal chest and you will obtain a clear, full sound which is called the pulmonary or vesicular resonance. Suppose one side of a chest to be partially occupied by a pleuritic exudation. If you then, with the patient erect, percuss that portion of the chest which is occupied by the contracted lung you will still obtain vesicular resonance, but it will be dull, as compared with the resonance of a healthy chest, or of the opposite unaffected side of the same chest.

If the percussion be applied directly over the part of the chest which contains the effusion, the sound will be destitute of resonance, and such condition of the percussion sound is called *flatness*.

Now dulness is a relative term indicating a diminution of resonance. Flatness is an absolute term indicating absence of resonance. However dull a contracted lung may be it is never flat so long as it possesses any resonance.

The dulness of the lung above a pleuritic exudation increases from above downwards, but the moment the percussion passes the line of demarcation between lung and effusion the sound becomes flat. Now this transition

from dulness to flatness is not a gradual gradation of one and the same quality of sound, but it is an actual well defined change of quality which is immediately perceptible to the ear and which can be delineated to the nicety of a hair's breadth. This change, to be sure, is more difficult to detect in some cases than in others, but one will rarely fail to find it if he percusses with proper delicacy and lightness.

If different parts of the chest be percussed from above downwards, and the points of transit from dulness to flatness be indicated by ink marks, these marks can subsequently be united by a continuous line which will accurately represent the line of division between that part of the chest still occupied by contracted lung and the part which contains the fluid exudation.

It will be remembered, however, that my remarks for the present apply to cases of fresh pleuritic exudations, uncomplicated by lung affections, or by any accidental adhesions, and once for all I announce that all cases of chronic pleurisy modified by extensive adhesions and all cases of circumscribed pleurisy, are referred to a later chapter for consideration.

The line of demarcation just mentioned is not a horizontal line such as would be obtained by percussing a drum which contains simply water and air, but it is a curved line and is called the

Curved Line of Flatness.

This line was first discovered by Damoiseau of Paris, and was subsequently rediscovered by Professor Ellis of Boston, who was the first to trace the true shape of the curve in its whole extent. Various other points concerning it, however, have remained very obscure.

Numerous opinions have prevailed as to the nature of the curve, and every author who mentions it seems to

have his own idea as to its shape, significance, and importance.

The object of this essay is to give a description of the true curve of flatness, to teach the proper way to search for it, to contribute certain experiments, which seem to throw some light upon the origin of the curve, and finally to discuss the diagnostic value of this much disputed symptom.

As described by Damoiseau, the pleuritic line of flatness is a parabola which is highest in the axillary region,

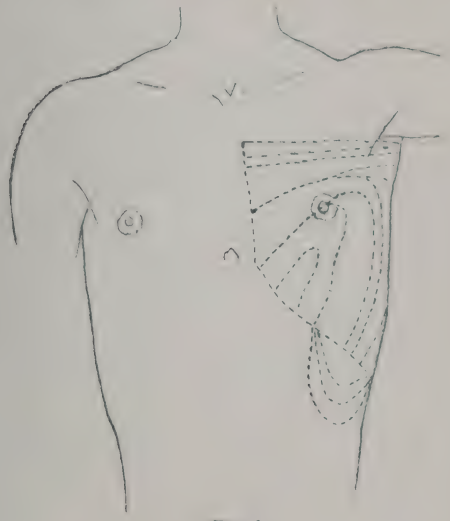


Fig. 1.

and which, under different circumstances, may present the various modifications exhibited in Figures 1 and 2. This parabola appears first in the axillary region, and as its summit ascends, its branches spread out in either direction toward sternum and vertebral column. Damoiseau concluded that a small effusion in the beginning of an attack of pleurisy ought to be detected in the axillary line before it would be perceptible anywhere else.

Wintrich describes a line of dulness (Dämpfung), which he says does not run parallel with the floor when the patient stands erect, but beginning at the vertebral column the line descends at an acute angle to the plane of the floor until it reaches the sternum. It sometimes presents undulations in the axillary region. He adds further that "often enough" the line assumes a parabolic character, as was correctly announced by Damoiseau and Hirtz, but he immediately modifies this concession by repeating his statement that the curve is always highest *behind*. Wintrich denies that a fluid exudation can be detected first in the axillary region, or that less than eight or ten ounces can be perceptible at all to percussion. According to him the fluid always appears first in the inferior dorsal region, and only when the fluid in this region has attained a height of three or four finger-breadths does the percussion sound become "shorter, duller, and tympanitic." This idea of the line of dulness, as described by Wintrich, has firmly established itself in Germany, and all German works reiterate it more or less mechanically. Fraentzel modifies the line somewhat, for he says that the line of dulness is highest behind and slopes down to the sternum with its concavity looking upwards when the patient assumes a half-reclining posture in bed.

Fig. 2.

Some German observers have obtained curves similar to Damoiseau, but they always hasten to explain such deviations from the Wintrich curve by assuming that the exceptional forms are due to accidental adhesions, or that they depend upon the fact that the patient has lain upon the affected side early and continuously.

I once heard Professor Huguenin demonstrate a case of pleurisy in the hospital at Zurich, when he drew upon the patient's chest a perfect curved line, but added, by way of comment, that such curves usually appear in the resolving stage, and are due to adhesions.

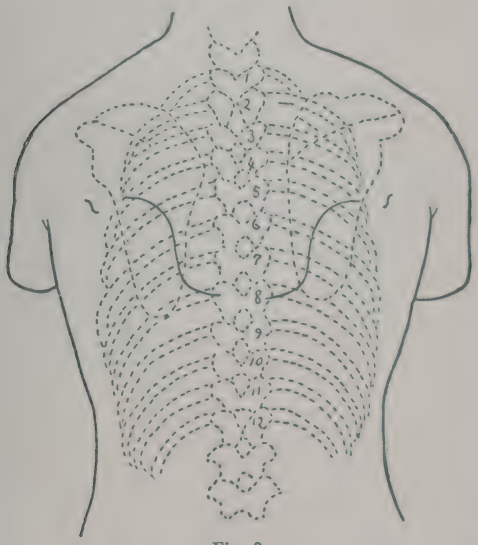


Fig. 3.

Anstie says that the dulness increases from below upwards, "but the line of its termination above is by no means always an evenly horizontal one."

Flint says: "If the trunk be in a vertical position, that is, the patient sitting or standing, the line of demarcation between the dulness or flatness and pulmonary

resonance is a horizontal line, on either the anterior, lateral, or posterior aspect of the chest."

These few quotations will suffice to show the general agreement among writers that a line of demarcation between lung and effusion actually exists, but they diverge most widely from each other in their opinions as to the position and shape of that line.

I take pleasure in referring to two papers published

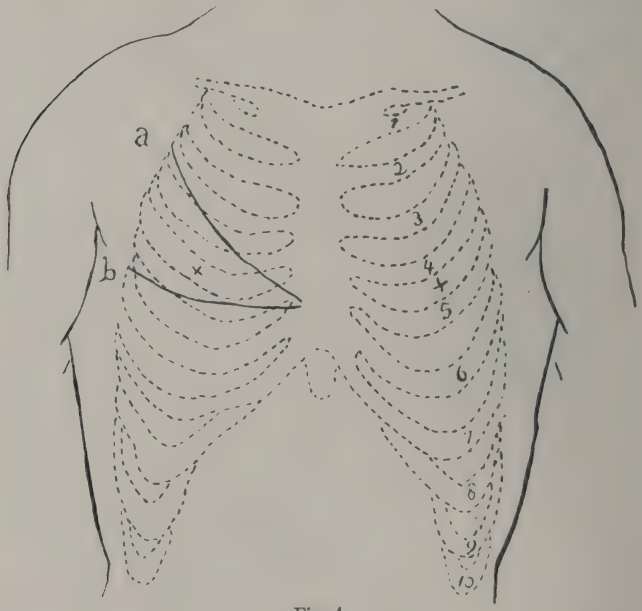


Fig. 4.

by Dr. Ellis, wherein the author demonstrates a line of demarcation between pulmonary resonance and effusion flatness, which is radically different both in shape and position from any line hitherto described. Dr. Ellis's line of flatness is a line which begins lowest behind, advances upwards and forwards in a letter S curve (see Figure 3) to the axillary region, whence it proceeds in a straight decline to the sternum.

A study of the Ellis curve will show that with small and medium effusions it retains the same general features, though the letter S is straighter in some cases than in others. With large and excessive effusions the curve undergoes certain modifications (see Case III.), but immediately returns to its original shape when the fluid is removed either by absorption or by thoracentesis. Moreover, the nature of the exudation has no effect upon the

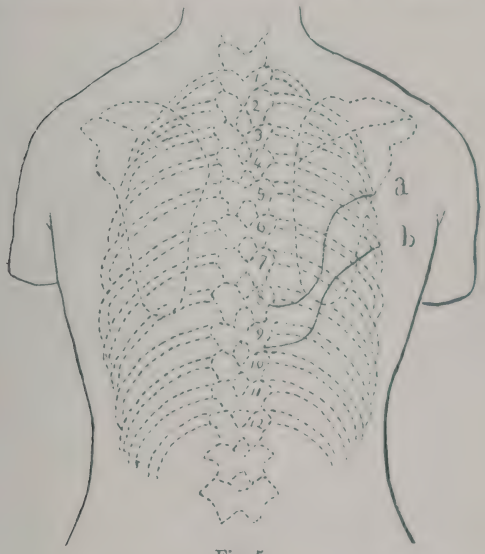


Fig. 5.

curve, for it is the same for pus as for serum. Compare Cases III., IV., and V.

The curve is never highest behind, even with the largest effusions, and the manner of its retrogression in the absorption stage is inversely the same as its development during the cumulative stage of the effusion.

The following is a short synopsis of Professor Ellis's cases.

CASE I. Presents no diagram.

Case II. An Irish currier, nineteen years old, entered the Massachusetts General Hospital on November 10, 1873. Was exposed to cold six weeks previously. Had a chill and pain in lower part of right side of chest. Was confined in bed for two weeks only. There was no cough or expectoration until two days before entrance

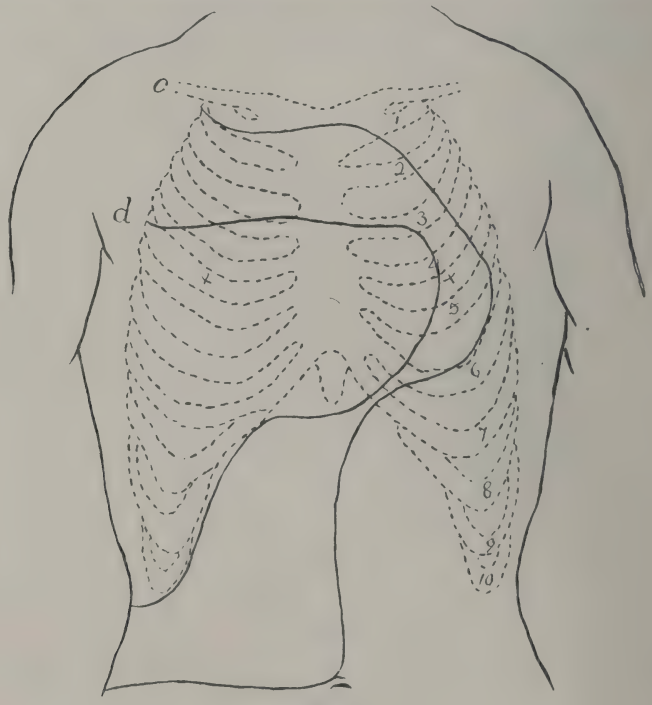


Fig. 6.

into hospital. There was flatness over the whole right side. On November 25th the condition of things was represented by the line *a* (see Figures 4 and 5), and the effusion gradually receded until December 28th, when the line *b* was drawn.

Case III. Native of Western Islands. No history

could be obtained, as patient did not speak English. The whole right side of chest was flat, except the top of the shoulder and the clavicular region. The heart was pushed to the left. (Figures 6 and 7, *c*.) Patient was tapped on October 4, 1873, between the eighth and ninth ribs, near the lower angle of the scapula, and eighty-four

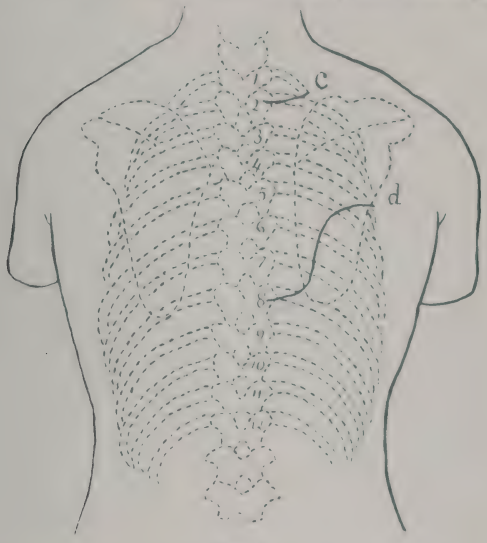


Fig. 7.

ounces of clear, yellow serum were drawn off. During the latter part of the operation there was much cough, which soon ceased. On November 2d the line of flatness was as shown in Figures 6 and 7, *d*. Above the line *d* respiration was heard everywhere, accompanied by moist râles on inspiration, and sibilant and sonorous râles on inspiration and expiration. On November 3d the flatness was the same, but the sibilant and sonorous râles had disappeared and the mucous râles were less abundant. Normal respiration was heard to the base along the spine, and from one to two inches outwards, the

area over which it was heard increasing towards the upper part, and following the line somewhat as indicated. Patient discharged December 15th.

Case IV. Hydrothorax. Boy with valvular disease of heart. On November 8, 1874, examination of the chest showed flatness of the right side below the line *e*,

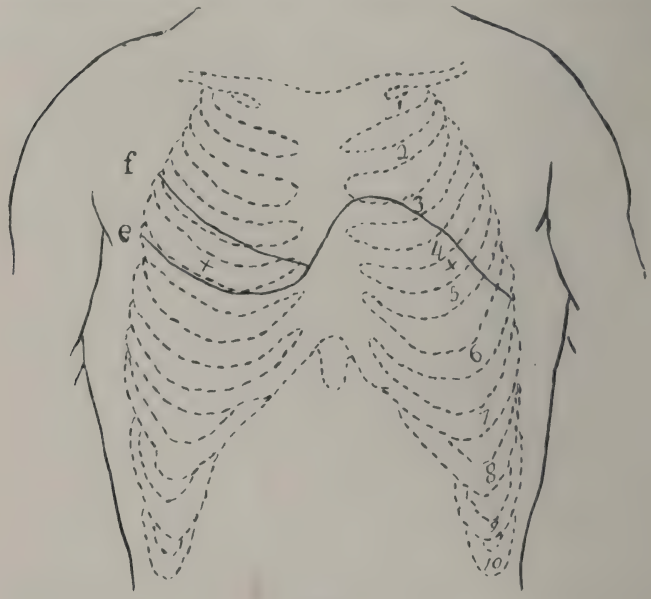


Fig. 8.

indicated in the diagram (Figures 8 and 9), the curve being best marked posteriorly. On November 16th the flatness had risen to the line *f*. Vesicular respiration was absent over a large portion of the flat region, but that of a bronchial character was heard over the lower third, commencing at the spine and gradually diminishing towards the post-axillary line, where it disappeared. There was also well-marked œgophony over this region. On November 24th the line of flatness had again fallen

below the line *e*. Respiration was heard even lower than the line, but ægophony persisted. Dr. Ellis adds, "This case is also interesting as illustrating the great rapidity with which fluid may increase and diminish. In judging of the efficacy of remedies this important point seems to be too frequently lost sight of."

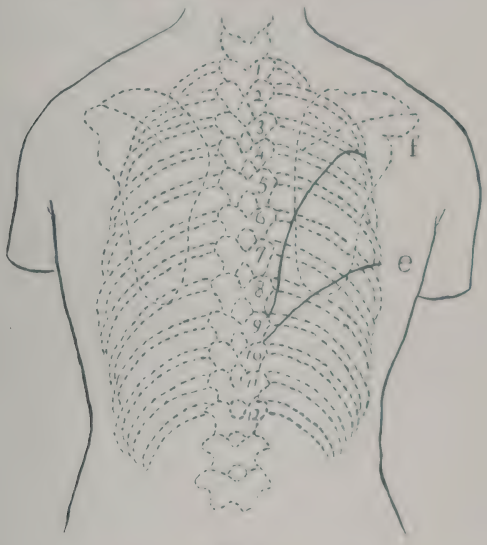


Fig. 9.

CASE V. Young man sixteen years old — service of Dr. Minot — reported that he took cold six weeks before entrance; had a chill; began to cough; perspired at night, and was troubled by pain in right side of chest. Three weeks before entrance he was tapped, and a pint and a half of pus was drawn off. On examination after entrance, the right side was flat below the line indicated. (Figures 10 and 11.) Introduction of a fine trocar proved that pus was still present.

"It is unnecessary to give the other physical signs, or speak farther of the case, as it is introduced here merely

to show the curved line as drawn by an independent observer."

In addition to these cases I have in my record-book the report of twelve cases which I examined in the hospitals of Vienna prior to the appearance of Dr. Ellis's second article. On comparing my curves with his, I find that my series corresponds with his in every essential detail, and therefore I do not think it will be necessary for me

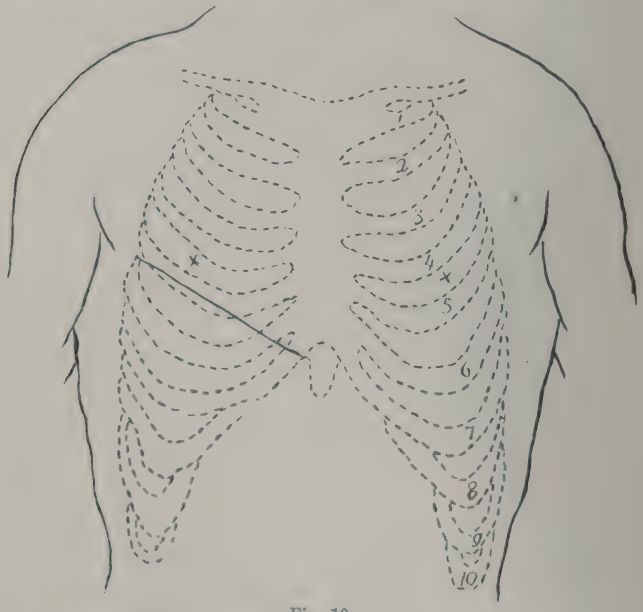


Fig. 10.

to multiply diagrams — I will only add that I examined one case of double-sided hydrothorax connected with valvular disease of the heart, in which I was able to trace the letter S curve on *both* sides of the thorax, and I also convinced others, who saw the case with me, of the accuracy of my percussion.

In order to readily detect the curved line of flatness,

it is necessary that the investigator should percuss in a correct manner, and according to certain rules. I will therefore indicate the manner in which I seek the line in question. In the first place, one should always percuss at right angles to the general direction of the curve, — the general direction of the curve is transverse across the chest, — hence, I always percuss from above downwards, in perpendicular lines.

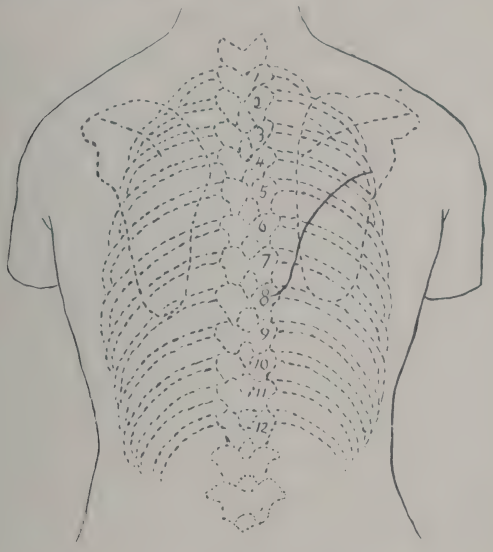


Fig. 11.

One should also proceed systematically from point to point on the chest, and as it is convenient to have certain definite lines of percussion for the sake of recording and comparing different curves, I have adopted the following schedule as a guide in my percussing: —

1. Vertebral line.
2. Dorsal line.
3. Posterior axillary line.
4. Axillary line.
5. Anterior axillary line.
6. Mammillary line.
7. Parasternal line.

The terms here employed are so suggestive and familiar that they will require no further explanation. For the sake of greater exactness in defining the lower part of the curve behind, I also percuss in a series of horizontal parallel lines running out from the vertebral column to the right or left as the case may be.

I must emphasize the necessity of percussing *lightly*, and I cannot urge this point too earnestly. It must be

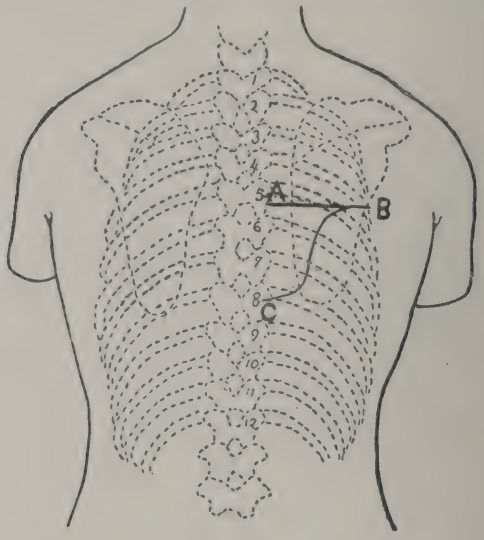


Fig. 12.

remembered that the body above the line is a resonant lung, while the body below the line is a non-resonant fluid, and therefore if the percussion near the upper edge of the effusion be strong, the flatness of the fluid will be entirely concealed by resonance transmitted from the lung.

One other rule is very essential to a successful percussion of the curve. In searching for the line of flatness one should *never* percuss alternate sides of the chest.

The point sought is not the distinction between dulness and full resonance, but between dulness and flatness, and as this distinction usually exists only upon the one side of the chest, the percussion must be absolutely confined to that side.

I do not say that one should *never* compare the two sides of the chest at all, but I do say, where the object is to trace the curved line of flatness, *Let alone the well side.*

It will be noticed that I have drawn upon Figure 12 the horizontal line A B, and that I have thereby enclosed an irregular triangular space which is bounded above by the line A B, on the side by the curve C B, and behind, by the vertebral column A C. This part of the chest is always the dullest portion outside of the flat area, and requires special delicacy in percussing. I have therefore termed it the "Dull Triangle," and its recognition is of the highest importance, as will be seen later. Hereafter, therefore, it will be understood when I speak of the dull triangle, that I refer to the portion of the chest which is shut off from the parts above by an imaginary line drawn from the summit of the curve perpendicularly to the vertebral column.

Do not understand me as saying that this horizontal line indicates any line of demarcation in the dulness. It is merely drawn for convenience sake, to call particular attention to a certain area of the chest where the percussion sound is often so very dull that it may be mistaken for flatness unless the percussion be very carefully made.

This is not always the case, however, and I will say, that, as a rule, the resonance of the dull triangle *ought* not to be mistaken for flatness.

I shall now proceed to describe certain experiments which, combined with the results of my clinical studies,

convince me that the Ellis curve of flatness is the only true curve when a pleuritic patient is properly percussed in the erect position, for I think that all the other curves mentioned are the result of improper distinction between dulness and flatness, or of certain accidental complications in the cases reported.

These experiments were undertaken with the view of discovering the actual relations between lung and effusion in a chest and in the hope that such knowledge might throw light upon the origin of the curve of flatness.

CHAPTER II.

EXPERIMENTS UPON DOGS.

THE first attempt to discover the explanation of the curved line of flatness by the introduction of fluid into the thorax of a dog was made by myself in 1874. I thought I might be able to study the relations existing between a lung and a pleuritic exudation if I could inject into the pleural cavity some fluid which by subsequent solidification would form a permanent cast of that cavity. I suspended the dogs perpendicularly by the head, and introduced a pear-shaped canula into the pleural cavity, by plunging it through the ninth or the tenth intercostal space in the axillary line. I employed glue and plaster of Paris for my injections, and exercised great care to prevent the entrance of air with the fluid.

Having allowed sufficient time for the solidification of the injection, I removed the animal's skin and percussed the chest, carefully marking upon the ribs the points of transition from pulmonary resonance to injection flatness. Then connecting these points by a continuous line, I obtained a curve of flatness which being lowest behind gradually rose to the axillary line, whence it proceeded nearly horizontally to the sternum, as shown in Figure 13. This curve was always the same in its general features for small and medium injections.

Little or no injection was present between lung and chest wall. The cast was always lowest behind and highest in the mediastinal region, where it often passed

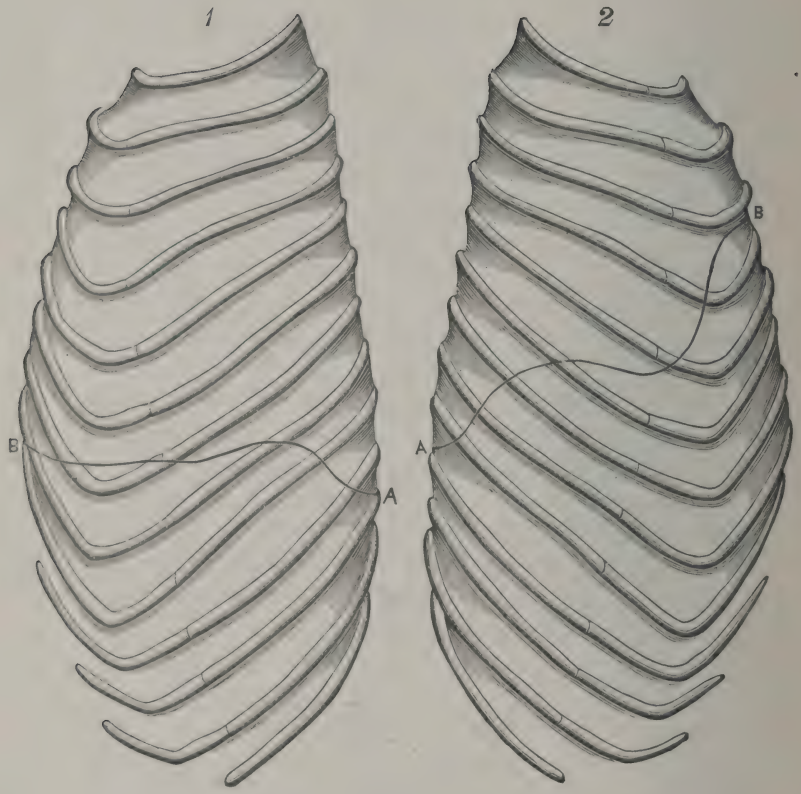


Fig. 13.

up around the heart. The fluid never assumed a hydrostatic level unless air had been accidentally admitted with the injection. The lungs were never *moulded* in any manner by the fluid, but preserved their normal form though reduced in volume. These results held good whether the injections were made into living or dead dogs.

During the following year my experiments were repeated by Dr. Ferber, of Marburg, who arrived at directly opposite conclusions. Ferber declares that plaster of Paris is not a suitable material for injection, because it solidifies too quickly, and for this reason he employed cocoa butter, which melts at 30° C. He also deemed it essential to inject into living animals. He concludes that "the position of the injection depends without doubt chiefly upon its specific gravity, and upon the position assumed by the animal during the experiment."

Although these, according to Ferber, are the chief factors, he recognizes that other conditions incidental to the experiment, and to the respiratory movements, as well as pathological casualties, may exert some influence upon the injection. I shall have occasion later to analyze the results obtained by Ferber, as he reports them, and therefore I will postpone for the present any criticism upon his conclusions. Prompted by Ferber's criticism I began a new series of experiments, which were at first conducted according to the latter's modifications.

The apparatus which I used for injecting consisted of a common wash-bottle, a canula, and a connecting tube of rubber. I filled the canula and tube with melted cocoa butter to exclude all air, and then plunged the canula into the ninth intercostal space and slowly injected. Large, medium, and small injections were made. The terms large, medium, and small, are relative, of course, to the size of each dog, as small injections for some dogs

would be large for others. I injected both living and dead animals, and placed them in various positions as the accompanying models will show.

When a medium injection was made with the dog in the perpendicular position, I subsequently obtained on percussion a curved line of flatness which was identical with that which I obtained three years previously. Then on carefully opening the chest I found that the external line of flatness invariably coincided exactly in position and curvature with the *lower border* of the lung, *i. e.*, with the *line of apposition* between the lung and the cast. This point is of the greatest importance and should be carefully borne in mind. I also found that the area of the dull triangle corresponded accurately in shape and position to the posterior inferior part of the lower lobe which still remained in *contact with the chest wall.*

The condition of things revealed on opening the chest was as follows: —

With small and medium injections in the vertical position the lung was diminished in volume but preserved its symmetry throughout. The lower part of the lung was not compressed to an airless condition and did not plunge into the fluid. On the contrary, the injection was situated beneath the lung and the latter appeared to rest upon the former. No injection was present between the lung and chest wall except an insignificant little ridge which never exceeded half an inch in height, and which was usually less than that. Moreover, with small and medium injections the diaphragm was arched strongly upwards and only a thin edge of the cast projected down into the complemental space.

During full expiration the diaphragm arches strongly upwards into the chest as represented by the curve A B C in Diagram 1 on opposite page. At the summit of full inspiration the diaphragm is flattened out by its own

muscular action and occupies the position A C. It is obvious, therefore, that with each act of inspiration the diaphragm increases the capacity of the thorax by the amount A B C. This additional room is immediately

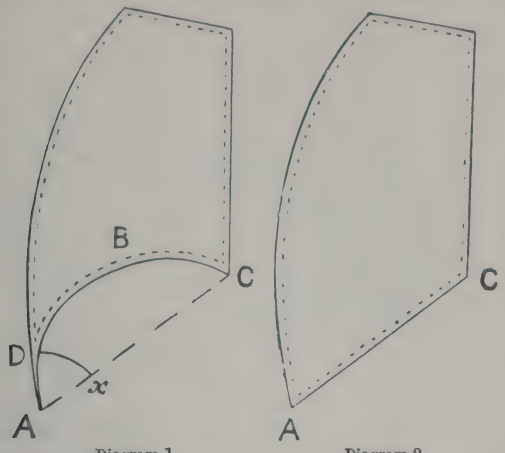


Diagram 1. Diagram 2.

occupied by the lower part of the lung, to be again abandoned during the expiratory retraction of that body. The space A B C, therefore constitutes, properly speaking, the true *complemental space* of the chest. Ordinarily, however, I think that term is applied only to a certain wedge-shaped portion of the above space, which I have indicated in the diagram by the letters D A X.

During full inspiration this space is occupied by the inferior posterior border of the lung, as represented by the dotted line in Diagram 2. At the end of expiration, however, this space is obliterated, so far as the thoracic cavity is concerned, by the apposition of the diaphragm to the chest wall from A to D, in Diagram 1. The dotted line in this figure indicates the contracted lung.

Of course the figures which I present above are intended to be purely diagrammatic and to represent simply the relation of the complemental space to the parts which surround it, and they, therefore, make no pretension to an exact representation of the actual configuration of that part of the chest or of its contents.

The conditions which prevailed after injections in other positions will be best understood by an analysis of the models obtained.

Model I.

Model I. Dog suspended by head in perpendicular position. Very small injection in right side — figure in plate is actual size by measurement. View from behind obliquely forwards. A B, portion in contact with vertebral column. A C D E F B, lateral surface, with impression of ribs. The superior border A C D was in apposition to the lower border of lung. The inferior border B F E D corresponds to the costo-diaphragmatic groove, and is very thin.

Broad surface A H D C A is a thin layer of fluid, which spread out between the lower surface of the lung and the diaphragm. It is thin as a sheet of paper, and is an exact cast of the lower surface of the lung, even to a reflexion of the minute pulmonary lobules which are represented in the plate by the dark spots at X. I need hardly add that there is no evidence of any hydrostatic level about this model.

Model II. Perpendicular position. View from behind, obliquely forwards. Medium injection in ninth intercostal space of left side. Outside surface of model is rounded, and corresponds to the curvature of the chest. It is also marked by impressions of ribs. Upper surface is *convex*, corresponding to concavity of lower surface of lung. Upper surface is also sharply inclined, being highest at the inside mediastinal edge, A, and lowest at the outside costal edge, B. The upper surface is also inclined from before backward. All of these inclinations correspond to the shape and position of the lower surface of the lung. The outer border of the upper surface of the model lay in immediate apposition to the lower outer border of the lung, and exactly represents the curvature of the latter.

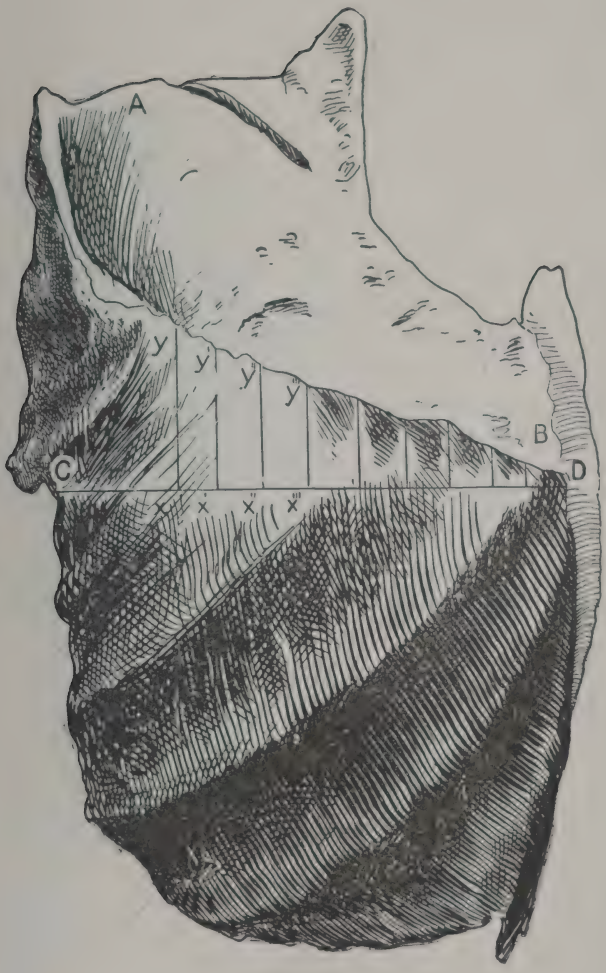
With injections which occupied less than one third of the thoracic cavity, there was scarcely a trace of a rim of the injection between the lower border of the lung and the chest wall.

The inferior surface of the model is concave, corresponding to the convexity of the diaphragm, and it bears the impress of the muscles of that membrane. The inner surface of the model exhibits grooves and depressions corresponding to the œsophagus, aorta, vertebral column, etc. The lower edge of the cast is thin as a knife-blade, and projected down into the complemental space. This model differs in no respect from one which I obtained by injecting a dog in the horizontal position, and immediately raising him to the perpendicular before solidification took place. It makes no difference when or how the dog be placed during injection if he be raised to the perpendicular position immediately thereafter.

On the reverse of this model is a groove which corresponds to the animal's vertebral column. Consequently, when this groove is held perpendicularly to the floor the

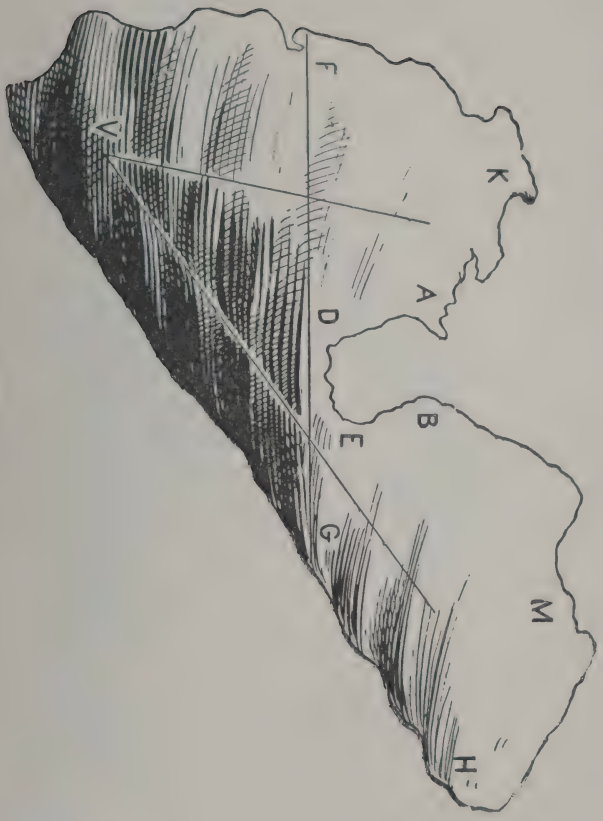
model occupies the position it did in the dog's chest because the dog's vertebral column was perpendicular to the floor, or practically so. I have drawn the line C D perpendicular to the vertebral groove behind, and consequently it is parallel to the floor.

The merest glance at the model will be sufficient to show that the line C D represents the imaginary hydrostatic level of all the fluid below it. I speak of *fluid*, because it will be remembered that at the time the adjustments represented in the model were made, the cocoa butter was in a fluid state. The youngest child, who had only studied the very elements of hydrostatics, would perceive that the body of fluid above the line C D does not present a hydrostatic level. On the contrary, the perpendicular lines, xy, $x'y'$, $x''y''$, $x'''y'''$, etc., represent columns of fluid which by some agency or other are elevated above the level which their specific gravity would give them, and the sum of all these lines represents a body of fluid sustained by some invisible agency above its natural level. Let us see if we obtain the same condition of affairs in our other models.



Model II.

MODEL III. Dog inclined at angle of about 45°. Large injection in ninth intercostal space of the left side. A thin portion of the model, corresponding to the small space A B D E, was destroyed in removing the mass from the chest. Now, if we draw the line F D G, we shall represent the hydrostatic level of the body of fluid below it. The two masses above that line, however, F K A, and E M G, are evidently in a state of inequilibrium which no principle of hydrostatics can explain. The blank space above K A B M was occupied by the lung, and the latter was still in *contact* with the chest wall.



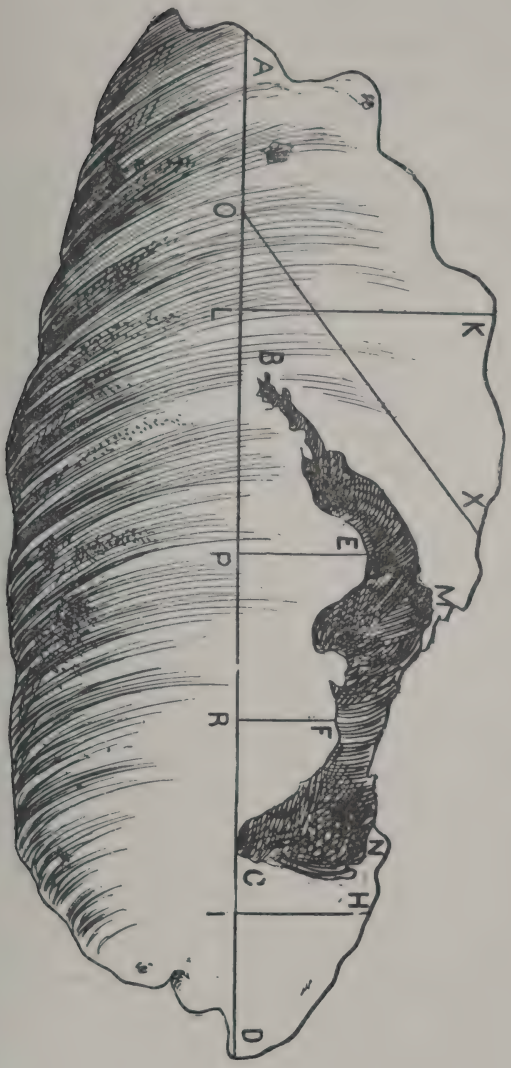
Model III.

Model IV. Dog horizontal. Very large injection. Here we have once more the same condition of affairs. Horizontal line A D represents the hydrostatic level of the fluid below it, but all the fluid above this line seems to have no possible means of support. B M N C is a large surface of the lung which was still in *contact with* the chest wall.

Now the question arises, What force supports the two columns K L and H I? If we have two flasks communicating at their base by a tube, and we pour water into one flask, it will pass through the connecting tube, until it stands at the same level in the second flask. One might say, therefore, that the columns K L and H I communicate at their base through the vertebral groove, and that they thus balance each other, though they cannot stand at just the same level, because of the slope of the chest wall. This explanation will not apply to our model, however, because if the two columns K L and H I were in a state of mutual balance, the columns E P and F R ought to be similarly related to those columns, since they also communicate at their base with the fluid in the vertebral groove.

We see, therefore, that one constant phenomenon is exhibited in all our casts. Large bodies of fluid are supported above their hydrostatic level by some agency not yet discovered. No principle of hydrostatics will explain this phenomenon. No change of position can affect it, since the condition of hydrostatic inequilibrium is constant in every position which the animal assumes.

In 1874 I concluded the account of my experiments, by the remark that the lungs seemed to be the moulding agent, and that they in some manner resisted the encroachments of the fluid. The irregularities of the upper part of the model were explained by the supposition that



Model IV.

the fluid spurted up in parts where the pulmonary resistance was least.

Maturer deliberation, however, convinced me that these conclusions were incorrect, because the lung is a non-resisting body, and therefore, cannot exert a counter pressure.

After considering the subject for some time, I decided that I could approach no nearer to a solution of the question by further experiments upon dogs. Accordingly, at Dr. Bowditch's suggestion, I turned my attention to a series of experiments which form the subject of the next chapter.

CHAPTER III.

EXPERIMENTS UPON ELASTIC BODIES IN ENCLOSED SPACES.

I TOOK a red rubber balloon, familiar to all as a child's toy, and having attached it to a glass tube, I suspended it in a pear-shaped flask by passing the tube through a rubber stopper. The flask has a second opening at its inferior apex, which is continued into a long nozzle B, and guarded by a revolving valve, C. (See Figure 14.)

On opening the valve and inflating the balloon all air is expelled from the flask, and the balloon accurately adapts itself to the inner surface of the glass like a coating of red paint. Then the valve C is closed, and the apparatus is ready for use. There is no valve in the glass tube above, and consequently the air in the interior of the balloon communicates freely through the tube with the external atmosphere. The balloon cannot contract, however, so long as the valve C remains closed below. Then the nozzle B is filled with water from a pipette, so as to exclude all air, and the flask is suspended over a beaker of water, with its nozzle dipping into the fluid beneath.

EXPERIMENT I. — I open the valve C. Immediately the balloon begins to contract, and is followed by a column of water, as represented in accompanying plate. The balloon does not collapse entirely, but soon comes to rest in the position photographed. If the flask be

plunged deeper into the reservoir below, the balloon again contracts. If the flask be raised higher, but with its nozzle still immersed in the fluid, the balloon expands again.

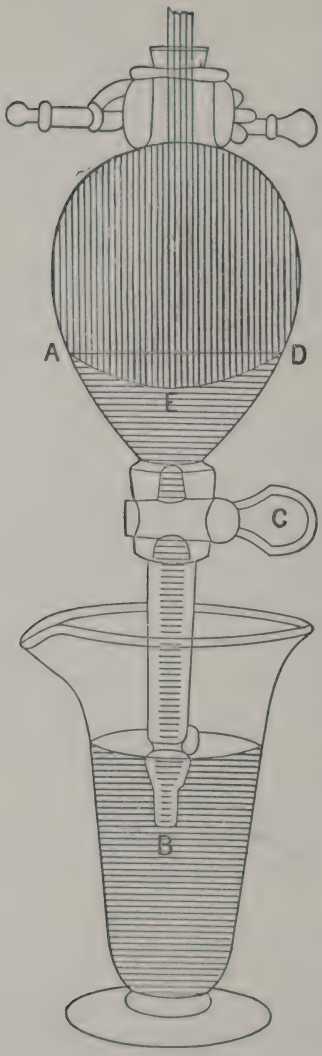


Fig. 14.

It follows, from this, that the retractile energy of the balloon is equivalent to the weight of a certain column of water, namely, E B ; and when this balance between the weight of the column and the retractile force of the balloon is attained, all motion ceases.

Further examination of our plate shows that the balloon appears to be plunged into the water. This appearance is purely an optical delusion, however, because the actual relation between the balloon and the water is just the reverse of that implied above. The balloon holds the water wrapped about it, so to speak, by virtue of its negative pressure. The column of water, instead of supporting the balloon, is actually suspended by virtue of the

retractile force of the latter. But if the column of water is in a state of suspension, it must exert a *negative* pressure upon the balloon by virtue of its weight. That it does exert such negative pressure is made obvious by raising the flask, and thereby lengthening the column of water. The balloon immediately expands.

But this is not all that our balloon teaches us. Notice once more that the portion of the balloon which is bathed by the water preserves a convex form, and that the upper surface of the water is correspondingly concave. How shall we explain this preservation of the shape of the balloon, and the conformation of the water to that shape? A moment's reflection will make these points clear. All solid bodies, of whatever shape, are held in form by the force of a so-called molecular attraction, which varies in intensity in different bodies. Those bodies most solid have the most powerful molecular attraction, and *vice versa*. For most bodies the force of this attraction is very limited in the sphere of its action, and consequently a very slight separation of the molecules in such bodies will result in their permanent severance. In one class of bodies called elastic, the sphere of the influence of molecular attraction is so extended as to admit of a certain amount of molecular motion without rupture of association. The play of the molecules within the limits of this sphere constitutes the phenomenon of elasticity. The more intense the force of the attraction the more elastic is the body said to be, while the more extended the sphere of attraction, the more extensible a body is said to be. The tendency of the molecular attraction of any body is to hold the molecules in a definite apposition, *i. e.*, to preserve a constant form for the body, and also to restore all sundered molecules to their former proximity to each other.

If an external force has separated the molecules from each other, but still within the limits of their mutual attraction, those molecules will constantly strive to reapproach each other, and this struggle increases in intensity according to the degree of separation. Should the limits of molecular attraction be passed, however, the magic spell is broken, the molecules become foreign to each other, and the elastic body ruptures.

As the external forces which separate the molecules always find them in a given relation to each other, and as the molecules always strive to return to the same relation, whatever be the direction of separation, it follows that the elasticity of a body always tends to restore the body to its original form, and the temporary shape which a body will assume under different degrees of distention will always be the balance of the antagonistic action between the conservative elasticity and the external distending forces.

Let us apply these reflections to our balloon by analyzing the different antagonistic forces which determine its shape. For convenience we will draw the line A D (see page 32). Now the forces which determine the shape of the balloon above the line A D are the retractility of the balloon, the atmospheric pressure within the balloon, and the molecular cohesiveness of the glass. So long as the valve C remains closed the retractility of the balloon is evidently unable to overcome the internal atmospheric pressure, or otherwise the balloon would contract in spite of that pressure and leave a vacuum behind in the flask. The glass itself is endowed with a very powerful molecular cohesiveness which the elasticity of the balloon is unable to disturb; consequently the balloon adapts itself to the form of the flask. If the flask were composed of some less inflexible substance, its external surface would show depressions here and there, which would correspond

to points of less resistance than the retractile force of the balloon.

Below the line A D the conditions of the antagonism are quite different. The forces which there come into play are the retractility of the balloon, the external atmospheric pressure, the internal atmospheric pressure, and the weight of the column of water. The mutual antagonism of these forces may be best illustrated by the following algebraic formula: —

Let x = Internal atmospheric pressure.
x' = External atmospheric pressure.
y = Retractility of balloon.
z = Weight of column of water.

Suppose, now, the external surface of the balloon to be the plane of meeting between the antagonistic forces. Then the force from above, when it arrives at that plane, will be the internal atmospheric pressure less the retractility of the balloon, *i. e.*, $(x-y)$. The force coming from below will be the external atmospheric pressure less the weight of the column of water, *i. e.*, $(x'-z)$. The difference between these two forces will determine the position of the balloon.

Thus $(x'-z)-(x-y)$ = Position of balloon.
But $x'=x$
Hence $x'-z-x+y=y-z$.

That is to say, the position which the balloon assumes is determined by the difference between the retractility of the balloon and the weight of the column of water. Supposing, now, instead of a column of water we substitute a column of air, then z will practically equal nothing and may be neglected. Our equation will then stand: —

$x'-(x-y)$ = Position of balloon.
But $x'-x+y=y$.

Hence, when air is let into the flask, the curve of the balloon will be determined by its elasticity alone. But this being true, the curve $y-z$ must differ from the curve y, and this difference is shown in the accompanying dia-

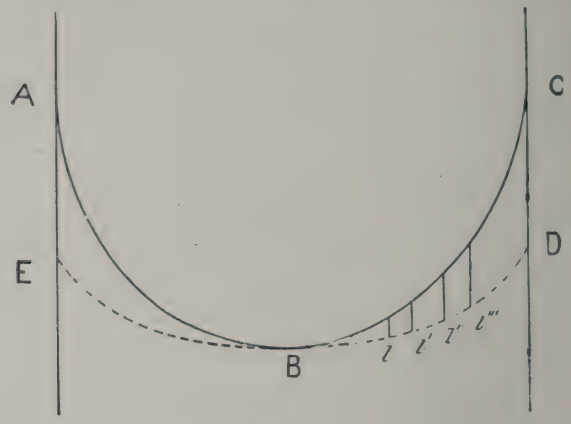


Fig. 15.

gram, which is drawn from curves obtained first with air and then with water.

A B C = curve y, *i. e.*, curve obtained with air in flask.

E B D = curve $y-z$, *i. e.*, the curve obtained with column of water, and the perpendicular lines l, l', l'', l''', etc., represent the negative pressure or traction of the column of water. The water holds the balloon back, so to speak. Fluids varying in specific gravity of course produce modifications in the shape of the balloon, corresponding to the difference in their weight, but I found that the difference in specific gravity must be very considerable, in order to produce any appreciable modification. We have analyzed, therefore, the forces which determine the position and shape of our balloon, and it only remains to explain the conformation of the water to the shape of the balloon. We have already seen that in the

upper part of the flask the balloon adapts itself to the glass, because the molecular cohesiveness of the former is less than that of the latter. The molecular cohesiveness of water is less than that of rubber, and hence the water conforms to the balloon.

One other experiment will further demonstrate the negative pressure of the water upon the balloon.

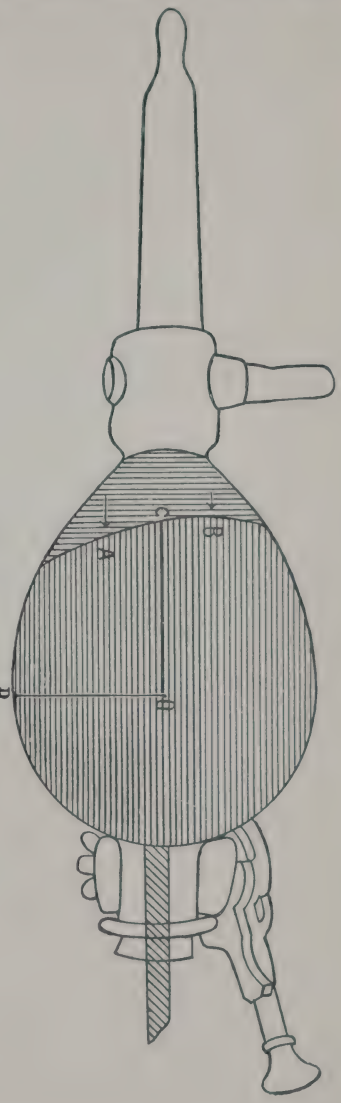


Fig. 16

EXPERIMENT II. If we close the valve C before the column of water has become equal in weight to the retractile force of the balloon, and if we then tip the flask upon its side, no change will occur between the water and the balloon. Place the flask perpendicularly once more and allow more water to enter. Again close the valve and recline the flask, and we shall obtain the relations exhibited in the horizontal plate.

Here we have a perpendicular column of water standing side by side, so to speak, with a retrac-

tile body which is striving to get away from it. The explanation of this seeming paradox is very simple. An elastic body retracts most strongly in those parts which are most distended. When an elastic balloon, therefore, is in a state of unequal expansion, it will retract with most force in those parts where its curvature is greatest, since those parts are evidently in a higher state of tension. Now, the curvature of our balloon is greater in the segment toward C than it is in the segment toward D. Hence the retractile force in the direction C O is greater than in the direction D O, and the fluid adjusts itself to the superior force. When, in the process of further retraction, the curvature toward C becomes equal to the curvature toward D, the fluid will pass under the balloon.

If we look carefully at the curve of the balloon, we shall notice that it bulges above the line C O, and is correspondingly flat below that line. Thus we have a different curve from any yet seen. The explanation is this: The water is now situated so that it exerts a lateral pressure upon the balloon. The pressure at the point B, however, is less than it is at A, since the lateral pressure of a layer of water is proportional to its depth. Therefore, the reaction of the balloon against the distending pressure of the internal atmosphere is more favored at A than it is at B, and hence the modification of the curve.

If milk be employed instead of water, in the above experiments, we can easily observe the action of capillary attraction by the thin layer of the milk which creeps in between the flask and the balloon. The layer is so thin, however, as to be almost colorless against the red background of the balloon.

Experiment III. Suppose, now, more water to be slowly driven into the flask from a syringe or by compressed air. The balloon will re-commence to contract,

and will, finally, if sufficient water be admitted, reduce itself to a state of complete collapse. Is this subsequent contraction due to any moulding influence of the injected water? Most certainly not.

We have seen that before the injection, the balloon supported the water. The injection was made slowly. The contraction of the balloon is very rapid. Hence it follows that the supply of water from behind is not so rapid as are the readjustments of the balloon in front, and consequently the balloon, so to speak, supports a column of water in advance of the influence of the injection.

Make the rapidity of the injection what you will, therefore, it is impossible that the advancing water should compress the fleeing balloon, until the velocity of the former exceeds that of the latter or until the elasticity of the latter is exhausted.

During the contraction of the balloon, however, its elasticity diminishes in intensity until it reaches zero, and in direct proportion thereto, the column of water supported by the elasticity diminishes until it also reaches zero. The zero point of the elasticity, therefore, is the point where all antagonism between water and balloon ceases. Now carry the injection one shade further, and compression of the already collapsed balloon begins, and may then be pushed to any extent desired by continuing the injection. It is obvious, therefore, that no fluid, under the conditions given, can ever compress a fleeing retractile body, until the elasticity of the latter is exhausted. It is also obvious that so long as the play of the elasticity of the balloon is unimpeded it can make no difference how the fluid is admitted into the flask. Whether it be driven in or be quietly drawn in by the balloon itself, as in Experiment I., the actual relations of the parts thereby established will always be the same. The individual steps of the changes which result will be

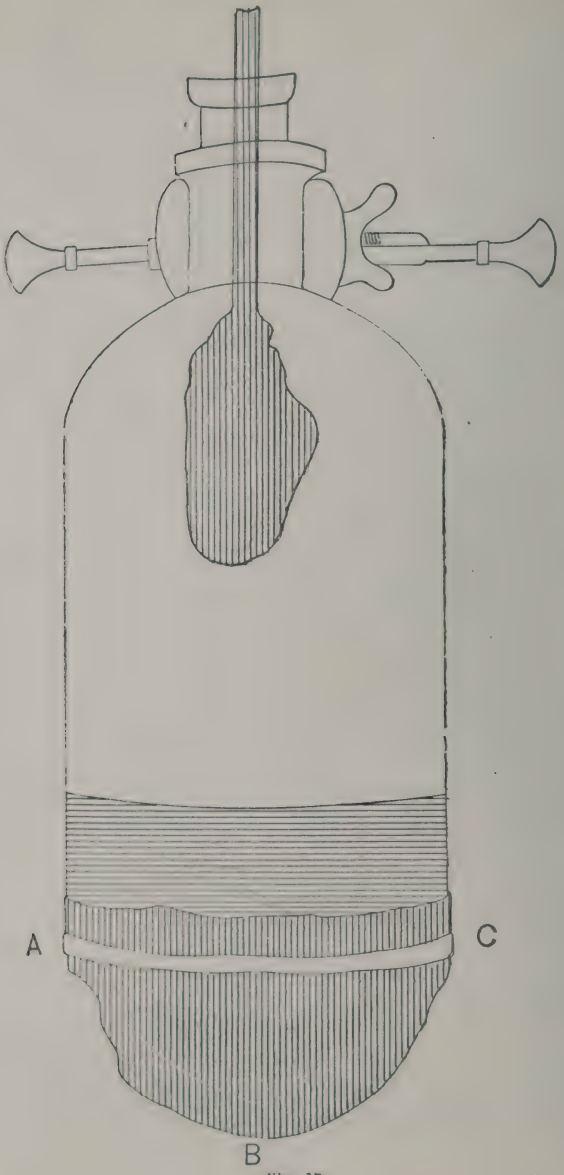


Fig 17.

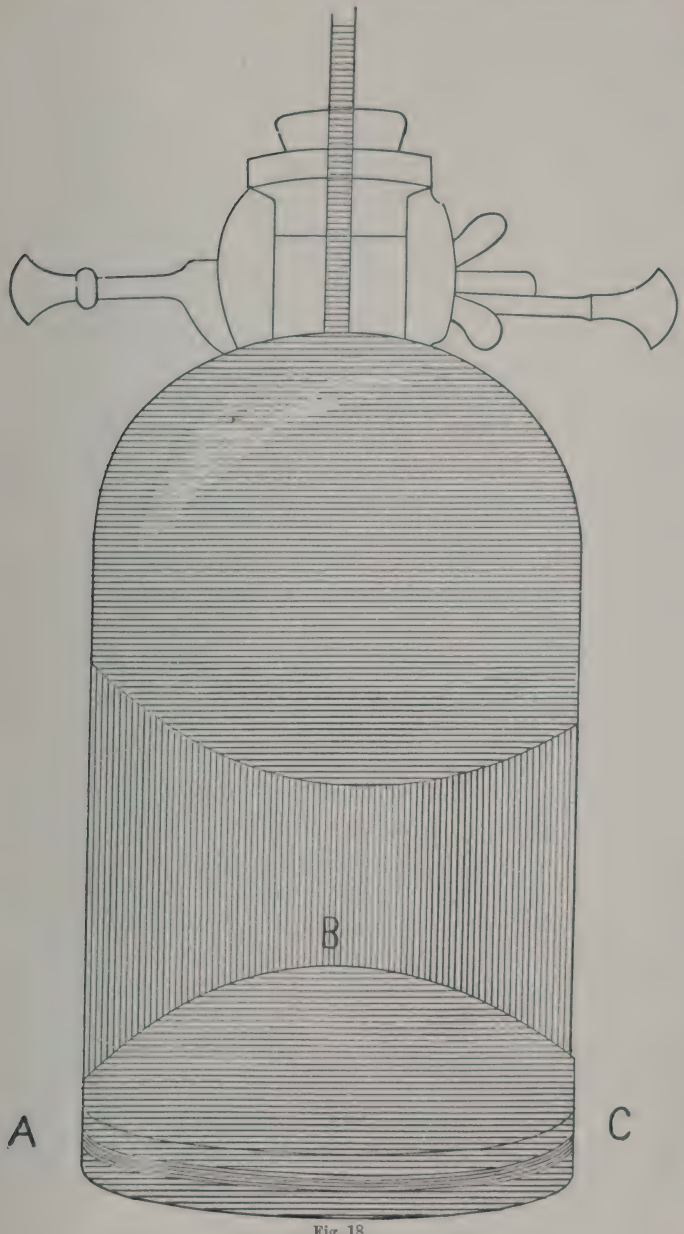


Fig. 18.

the same. The velocity of their succession will merely be accelerated.

EXPERIMENT IV. Suppose, now, we take a bottle, and having removed the bottom of the same, substitute therefor a rubber membrane. Then partially fill the bottle with water, and suspend therein the balloon as in first experiment. The weight of the water will depress the rubber membrane so that it will arch downwards, forming the curve A B C on page 40. If the balloon be now inflated until all air is expelled from the bottle, and if the stopper be then accurately adjusted and the balloon be allowed to contract by opening the escape tube above, the result will be that seen in the plate on page 41.

The rubber membrane no longer bulges down, but is strongly arched upward and considerable force is necessary to pull it down from beneath.

In this case both the water and the rubber membrane are lifted up by the negative pressure of the balloon. As the membrane is raised, however, it becomes tense and resistant. Now, the tensile resistance of the membrane is proportionate to the degree of its convexity, and hence a point is soon reached where this resistance, plus the weight of the water, becomes equal to the lifting force of the balloon. At this point, therefore, equilibrium is established and motion ceases, and this is indeed the condition of things represented in Figure 18.

We have seen, by Experiments I. and II. of this chapter, that the water exerts a negative pressure downward upon the balloon by virtue of its weight. It is evident that the water also exerts a direct pressure downward upon the membrane. Why does it not depress the membrane? I have just shown that the lifting force of the balloon is superior to the weight of the water, since it is equal to that weight, *plus* the tensile resistance of the membrane below. It follows, therefore,

that the membrane is supported by a force which is superior to the weight of the water, and hence the water cannot depress it. Suppose, now, the maximum lifting force of the balloon has been attained, and we add a larger amount of water. We shall thereby produce an excess of weight of water, and this excess will depress the membrane until finally the latter will bag down again as in Figure 17. Moreover, since this excess of water acts most powerfully where it is deepest, the depression of the membrane will appear first along the line of its attachment to the bottle.

A few more words are necessary with regard to the lateral displacing force of the water. If the lower layers of the water, which transmit to the membrane below the pressure acquired from superincumbent layers, are unable to produce a downward displacement of that membrane, they will be equally unable to transmit an efficient lateral displacing force against the sides of the bottle. Hence, if the sides of the bottle were likewise composed of rubber they would convex inwards in the same way that the membrane arches upwards. It follows, therefore, for reasons given, that no obliteration of the inward convexity of the sides of the bottle would be possible until an excess of water was present. Hence, *ceteris paribus*, the beginning of the obliteration of the inward convexity of the lateral walls would be simultaneous with the beginning of the depression of the membrane below. It is also evident that this obliteration would appear first in the lowest part of the bottle, since the lowest layers of a fluid always transmit the greatest amount of lateral pressure.

The principles thus far developed are of universal application to retractile bodies enclosed in firm walls, and hence may be, with propriety, applied to the retractile lung in the thorax.

CHAPTER IV.

ANALOGY BETWEEN DOG'S LUNGS AND ELASTIC BODIES IN ENCLOSED SPACES.

A DOG'S lung is a highly elastic body shut up in an enclosed space, namely, the thoracic cavity. Under normal conditions this lung is always distended beyond the zero point of its elasticity, and hence there is a perpetual struggle within the chest between this elasticity and opposing forces. The typical form of the lung is that assumed in the condition of complete collapse, and the force of the elasticity is expended in an incessant endeavor to restore the lung to that form. It must be true, therefore, of the lung, as of the balloon, that the form which it will present, at any given stage of distention, will be the result of the antagonism between its elasticity and all external forces operating against it.

The forces ordinarily operating against the pulmonary elasticity are, chiefly, the action of the external respiratory muscles and of the diaphragm, the inflexible nature of the chest walls, and the atmospheric pressure. If we make an injection of fluid into the thoracic cavity, we add one more factor to the opposition, and that factor is the weight of the fluid injected.

The general conclusion, towards which my argument is tending, seems so obvious now that it hardly needs to be stated.

The elasticity of the lung, however strong or however weak it may be, is equivalent to the weight of some column of fluid.

It follows, therefore, that when a fluid is subjected to the action of that elasticity, it will be raised above its hydrostatic level and assume a pneumono-dynamic level, if I may be allowed the expression, and the amount of fluid raised will be equivalent to the energy of the pulmonic retracting force which raises it. We saw that the balloon not only lifted a column of fluid, but it distributed that fluid according to its own form, and according to the relative force of its retractility in different parts. Theoretically the same must be true of the lung, and the fluid raised must be distributed according to the shape of the lung and according to the relative force of its retractility in different parts. These are general statements of theory, however, and we must now see if they will bear the test of actual application to facts.

We discovered on analyzing our models that they presented certain anomalies which we were unable to explain. We found certain columns of the fluid which shot up above the surrounding body of the injection, and which were there maintained by some force unknown to us. Moreover, we noticed that the under surface of each of our models was concave, corresponding to the convexity of the diaphragm when arched into the thorax. It follows, therefore, that the diaphragm and the injection were both drawn up into the thorax by the lifting force of the lung. In short, we discover, by our models, that the whole condition of affairs in the chest is exactly analogous to the various conditions analyzed in the preceding chapter, and the key to the interpretation of one and all of these phenomena is the

Elasticity of the Lung.

The columns of fluid which we have seen in our models represent the distributing energy of the lung, and the conformation of the fluid to the shape of the lung.

They by no means represent the whole retractile energy of the lung, however, because we have also seen that where the diaphragm remained convexed upward, as it was found on opening the chest, the entire weight of the injection was still less than the lifting force of the lung. Now Model II., page 25, represents an injection which filled about one third of the chest, and was made with the dog suspended perpendicularly, and yet the diaphragm was not bagged down. This will afford us some idea of the retractile force of the lung, though I have not as yet made any direct experiments to determine this point more definitely. I noticed, however, that, with larger injections, the diaphragm began to bag, and that this bagging appeared first behind, and then advanced up the side along the costal attachments of the membrane. It is obvious, therefore, that when the entire weight of the fluid begins to exceed the elastic force of the lung, the excess of weight begins to depress the diaphragm. This excess of weight will make itself evident first in the thickest part of the injection, and that is below and behind. Of course, when the dog lies horizontally, the excess of the injection will not bear directly upon the diaphragm, and hence very large injections may be present in the horizontal position without any depression of the membrane, and examination of my models proves this to be the case. I imagine that this depression ought to be greater on the right side than on the left, because it will be aided there by the negative pressure of the weight of the liver below; but I have made no experiments bearing upon this point.

Negative Pressure of the Injection. — It is a self-evident corollary of what has preceded, that the suspended fluid can *not compress* the lung which suspends it. On the contrary, it must have an opposite effect, and by virtue of its weight, it must exert a negative pressure

upon the lung. We have seen (page 36), the difference in the curves which the balloon presents when retracting before a volume of air and before the same volume of water. The fluid injection in the chest must exert an analogous restraining influence upon the lung.

The rapidity and force of the injection can make no difference. Even if the rapidity of the adjustments of the lung is not so great as the rapidity of the injection, yet, the amount of the fluid is so limited, that after the injection ceases, the contraction of the lung will proceed until the ultimate conditions of affairs will be that represented. The locality of the injection, and the position of the animal at the moment of the injection, do not alter the results.

I have injected dogs in the horizontal position and then immediately placed them erect before the fluid was hardened, and I obtained exactly the same results as when the injection was made primarily in the erect position.

In Model II., page 25, we notice that no injection is present between lung and chest wall, or, at best, only a small rim of fluid, which is very insignificant in amount. In Model IV., page 29, we perceive that a thin layer of fluid, B E F C, lies between the lateral aspect of the lung and the chest wall. Let us examine, therefore, the conditions which prevail in the two cases, in order to discover why the fluid is present between the lung and chest wall in the one case and not in the other.

In Model II. it was evident that the entire body of the injection was suspended, so to speak, from the lung, and as no fluid can rise above its highest point of support, of course none of the injection could penetrate between lung and chest wall. But one may object that the lung contracts laterally as well as perpendicularly, and should thus draw the fluid up. Experiment II., on page 37, taught us, however, that a contractile body retracts with

most force in the radii of its greatest distention, and that the fluid adjusts itself to the superior force. The lungs are most distended in their lower part, and hence the vigor of the retraction of that part and the consequent adjustment of the fluid.

One might object, again, that the movement of the ribs would tend to draw fluid up between themselves and the lung. When the ribs are elevated and the chest thereby expanded, a potential vacuum forms between the ribs and lung. Why does not the injection rush in to fill this vacuum, since the weight of a small amount of fluid is less than the elasticity of the lung? It must be remembered, however, that the elasticity of the lung is not greater than the weight of the atmospheric pressure. When, therefore, the ribs, by their action, lift off the external atmospheric pressure, the air within the lung immediately rushes in to fill the vacuum and sweeps the lung along with it. Hence the relative weight of the fluid cannot be affected by the movements of the chest wall. In speaking of a "potential vacuum," I do not, of course, mean that an actual vacuum of large size is formed and subsequently filled. The retirement of the ribs and the pursuit of the lung must be simultaneous.

The body of fluid, OX, Model III., page 27, forms the most striking feature of any of our models. It presents to us a seemingly double paradox. First, we have a body of fluid projected above its hydrostatic level, and secondly, we have a column of liquid inclining at an angle, like the Leaning Tower of Pisa, and without visible means of support. The lung beneath cannot brace it up, since that organ is striving to retract in the opposite direction.

And yet the explanation of this is very simple and has already been thoroughly demonstrated in connection with the horizontal flask, page 37. We there saw that

the lower part of the balloon was able to support a perpendicular column of water beside it by virtue of the superiority of the retractile force in that part. The same is true of the lung when the dog is placed upon his back. There is this difference, however, between our horizontal flask and our dog's chest. In the former the wall behind the water was composed of inflexible glass. In the dog's chest the wall behind the cocoa butter is the flexible diaphragm. The diaphragm yields to the elasticity of the lung, and consequently the lung retracts relatively further than the balloon was able to do. The result of this is that the perpendicular column of water in the one case becomes a leaning column of injection in the second case. When the diaphragm was drawn up, however, as high as possible, it refused to yield further, and thus checked the inclination of the fluid at the critical point where we see it.

It will further be seen, on Model IV., page 29, that in the horizontal position, with a very large injection, the retractile force of the lower part of the lung supports a large body of fluid A K M B while the lateral retraction only supports the thin layer B E F C. This again is to be interpreted by the principle evolved from the horizontal flask, namely, that the injection is distributed according to the balance of retractility in different parts of the lung.

Compression of the Lung. — I have said that the water cannot compress the balloon until the retractility of the latter is exhausted. The same must be true of the injection and the lung. Moreover, so long as the diaphragm is arched upward like the membrane in Figure 18, it can offer no point of resistance to the injection and therefore the latter will be unable to compress the lung upward until the diaphragm is arched downward.

Capillary Attraction. — The exceedingly thin

layer of fluid which can be supported by capillary attraction under the most favorable circumstances is so insignificant as compared with the large forces and weights which we are at present considering that it may be practically ignored. I have spoken of a narrow, thin rim of fluid along the upper border of my perpendicular model. This may be due to capillary attraction, but I think its weight is too great to be thus accounted for. I should rather assume that the lateral retraction of the border of the lung was just sufficient for this narrow rim and hence it was drawn up. It is too small, however, to have any appreciable effect upon the percussion sound, and this brings us to the consideration of our curved line of flatness.

Curved Line. — The true letter S curve of flatness, as described in Chapter II., is observed only when the dog is retained in the perpendicular position, and I have shown that on opening the chest it was found to correspond accurately to the *line of apposition between the lower border of the lung and upper rim of the injection.* As the shape of the lung determines the shape of the upper border of the fluid, so it determines the shape of the curve, which is simply the letter S curvature of the lower border of the lung, and the curvature of that border for any given amount of injection is the result, of course, of the balance between the elasticity of the lung and the weight of the injection. It will be noticed, however, in Model IV., page 29, that the anterior portion of the curve, M B, still persists even in the horizontal position.

Distribution of an Injection. — A study of all my models shows that the distribution of the fluid in the chest follows one general plan in all the positions indicated.

Model I., page 22, which is actual size, shows us that

in the vertical position the fluid collects first in the lower posterior part of the pleural cavity, *i. e.*, in the complemental space. Thence it extends in a narrow band upwards and forwards between the lower border of the lung and the costal attachments of the diaphragm. At the same time it spreads out in a thin sheet over the diaphragm. The further development of the injection proceeds in exactly the same manner until the fluid has attained the thickness and height of Model II., page 25. Beyond this point the injection begins to bag down the diaphragm, to obliterate the intercostal depressions, and to rise behind the lung in the vertebral groove, and lastly it appears between the lung and lateral chest wall.

When the dog reclines, or lies horizontally on his back, the injection collects below and behind in the complemental space, and thence extends up over the diaphragm, and along the vertebral groove. The two directions of advance, therefore, form a letter V, with its apex in the complemental space below. (See Model III., p. 27.)

Having ascended the vertebral groove to the top of the chest, it is drawn up alongside the apex (see Model IV., p. 29), and *finally* appears in a thin sheet between lung and lateral wall of chest. The diaphragm does not bag down in any case until the entire weight of the injection exceeds the supporting force of the lung. Moreover, the injection can exert no efficient lateral displacing force until the elasticity of the lung is no longer sufficient to support the entire weight of the fluid, and hence the intercostal depressions will not become obliterated until the diaphragm bags down.

One more fact is worthy of note in connection with the curve. I employed in my injections different fluids of varying specific gravity, such as water, plaster of Paris, glue, and melted cocoa butter, and I always obtained

practically the same curve on the chest, and the same internal relations.

Recognizing the fact, therefore, that the shape of the lung must be in part due to the negative pressure of the injection, we nevertheless perceive that fluids which differ only slightly in specific gravity will produce only inappreciable modifications in the shape of the lung when injected into the chest, and therefore we shall *never* be able to judge from the *shape* of the curve on the chest as to the *nature* of the fluid within.

Note. — All the models described in Chapter II. were obtained, as I have stated, by injecting the pleural cavity. The injection of the fluid seemed necessary, for two reasons. In the first place, I employed a very small canula to avoid the entrance of air, and hence the friction was great, and retarded the flow. Moreover, in the beginning of my experiments I used glue, plaster of Paris, and paraffine; and as these substances solidify with considerable rapidity, it was necessary to hurry their admission into the thorax. When I adopted cocoa butter for my injections, I found that it solidified so slowly that a hurried injection was unnecessary. Accordingly I prepared a large glass canula of pear shape, and attached it to a rubber tube. Having filled the canula and tube with the melted butter, to the exclusion of all air, I plunged the instrument into the pleural cavity, and immersed the outer end of the tube in a basin of butter. The basin was placed at a slightly lower level than the point where the canula was inserted.

As a result of this experiment I found that the elastic force of the lung is not only able to support a large body of fluid in the chest, but it is also able to draw up a large body of fluid into the chest. It seems to me that this is a crucial test, and conclusive proof of the correctness of my theory.

The last experiment of this kind which I performed illustrated most beautifully the principles which, I have said, govern the distribution of the fluid in the chest. I placed the dog etherized, upon his right side. The animal was living, and lay

breathing quietly, as if asleep. The canula was introduced in the axillary line of the left side, which was uppermost. After sufficient fluid was drawn in, and time had been allowed for adjustments, the dog was killed. On opening the chest, the following conditions were discovered. The thorax contained a large amount of solidified butter which was distributed in both cavities, since fluid will readily filter through the mediastinum of a dog. On the left side, which was uppermost, there was absolutely *not a drop* of fluid between the lateral surface of the lung and the chest wall. A mass of butter lay between the lung and diaphragm, and another mass projected up the vertebral groove, thus forming a typical letter V, such as I depict on Model III., page 27. The main body of the fluid lay like a saddle over the heart, and seemed to be balanced on either side by the mutual antagonism of the two lungs.

In the lower half of the chest I found a large mass between the lung and diaphragm, and a small amount in the vertebral groove. Now, it is evident that the dog's right chest wall was the lowest part of his thorax, since he lay on that side. Hence, simple gravitation would have brought down the fluid to that region, and one would naturally expect to find a large quantity of this between the lateral surface of that lung and the costal pleura. On the contrary, however, there was merely the thinnest, wafer-like bit of butter about the area of a silver dollar, in that region. The lung lay in *apposition* to the chest wall.

It seems to me that nothing could be more conclusive of the correctness of my theory regarding the distribution of fluid in the chest than was this experiment. Each lung, irrespective of its position, one above and one below, had appropriated about an equal amount of fluid, and both had distributed the same alike, since the balance of the elasticity of their different parts was similar.

Hence I conclude that the essential conditions for all our models are: —

I. Presence of fluid in the pleural cavity.

II. Lifting force of lung.

III. Balance between the retractile energy of different parts of the lung.

V. Position of the body.

IV. Amount of the fluid.

VI. Antagonism between the two lungs.

The variations in the retractile energy of different parts of the lung, are of coursé determined by variations in the distention of those parts, as I have explained in the case of the balloon.

CHAPTER V.

CRITICISM UPON FERBER.

In Chapter II., I said that Ferber had arrived at opposite conclusions to my own. He asserted that the form of my early models was due to the rapid solidification of the material injected. In reply, I will say that in my recent experiments I employed cocoa butter, and I obtained the same shape of models as with plaster of Paris. Moreover, on carefully rereading Ferber's descriptions of his own models, I find that they agree more nearly with my own than he seems willing to allow.

An analysis of his four models, as reported, will make this point clearer.

"Model I. Obtained with the dog lying upon his back. The fluid lay along the vertebral column and showed *no depression* of the diaphragm. The external, upper border presented slight pointed projections and ran *nearly* parallel with the axillary line, that is, nearly horizontal. Injection through the ninth intercostal space in the axillary line of the right side. Large dog."

The italics in this, and in the other quotations of this chapter are my own. Now a fluid whose upper edge runs "*nearly*" horizontal, and presents even slight pointed projections, can scarcely be said to have assumed a hydrostatic level. No fluid can change its level voluntarily. However slight may be the deviation of its surface from a horizontal level, that deviation indicates the inter-

ference of some external agency. It will also be noticed that the diaphragm was *not* depressed.

"MODEL II. Injection through the ninth intercostal space in the axillary line of the right side. Position obliquely elevated as a man lies in bed. This model corresponds precisely with the line of dulness on men, which generally descends from a higher point on the back to a lower point in front. The major part of the model collected in greatest thickness near the lower dorsal vertebræ. The surface of the diaphragm was only covered in part. The anterior superior portion was bare. The upper outer border was horizontal for the position assumed during the experiment, and when the animal was lifted up, it descended in a slightly wavy line from behind forwards."

It will be seen that this model, though very imperfectly described, corresponds in the main with my Model III., and hence the explanation of my model will serve as a criticism upon this one of Ferber.

"MODEL III. Injection under water, through the second intercostal space in the mammillary line of the right side. Position almost vertical upon the hind legs. The exudation rested in a thick layer upon the diaphragm. One can see the posterior complemental space of the pleura plainly marked upon the model. The superior surface of the model is *convex*, corresponding to the *concavity* of the under surface of the lung. A very thin layer of exudation, extending from the front backwards, rose to the height of two finger-breadths between the inferior lateral border of the lung and the thorax wall."

I need hardly call attention to the fact that a fluid whose superior surface is *convex* is not in a condition of hydrostatic equilibrium, and yet Ferber glances off from this important fact, as if a convex surface were the

normal condition of a fluid. The description of Model III., however, is imperfect, because it does not contain one very important point which Ferber mentions in an incidental manner three pages later. He there says, "If the exudation lies upon the diaphragm, then the impression of the lung on the model is certainly *lowest* behind and thence *rises forward*." It will be seen, therefore, that Ferber's Model III. with its superior convex surface, which slopes from the mediastinal region downwards and backwards, is exactly the counterpart of my Model II., page 25.

No further evidence can be needed to prove that the results which Ferber obtained are identical with those which I obtained and have described. Ferber's thoughts, however, seem to be riveted upon one idea, namely, that the body of the injection seeks and occupies the lowest points of the chest, and he utterly fails to appreciate the significance of the upper part of a fluid being maintained by an invisible support, in a condition of hydrostatic inequilibrium.

His fourth model is described as follows: —

"Model IV. — Injection in the axillary line of the right intercostal space. Position perpendicular upon head. The exudation sat like a hood upon the apex of the right lung, and was limited by a superior horizontal line."

I made no injection under the conditions mentioned with this model, but the results obtained are precisely what I should expect from the principles which I have already explained. The apex of the lung is not concave, like the base, but it is convex, like the balloon, and hence the mutual adjustments of apex and injection coincide with those between balloon and water in Experiment I., Chapter III. If the water in the flask should solidify the model would appear like a hood upon the balloon.

Moreover, its upper border is horizontal, and it is horizontal because the line of cleavage between the balloon and the glass is horizontal, and not because the specific gravity of the water places it so. The superior surface of the water, however, — and this is the striking feature of the experiment, — is *concave*. The superior surface of Ferber's model was also *concave*, because Ferber says it rested upon the apex *like a hood*. How can a fluid, whose superior surface is concave, be in a state of stable equilibrium?

Since writing the above I have made two injections into the thorax of dogs which were suspended vertically, head downwards, and I obtained conditions similar to those described by Ferber. I detected, however, a complication in the experiment, which exerted a marked influence upon the result. Before suspending the dog by his legs, I made a small opening in the abdominal wall for the insertion of my finger, which I always passed up under the ribs, in all my experiments, in order to control the introduction of the canula.

After the dog was hung up, I found that the diaphragm, which had been previously accessible in every part to my finger, was now so far retracted into the thoracic cavity that I could no longer touch it in the centre.

The explanation of this condition and the results which it would entail were immediately clear to me. By inverting the animal I had caused the heavy weights of the liver and stomach to bear directly down upon the diaphragm and hence it was convexed into the chest to a very abnormal degree. Moreover, these weights, in depressing the diaphragm, had allowed the lower part of the lung to retract further than normal. It was obvious, therefore, that the superior retractile energy of the lower part of the lung was so diminished by reason of this contraction, that the injection would be free to gravitate to other parts of the chest, and this I found to be the result.

The striking coincidence between Ferber's results and my own, therefore, is too self-evident for doubt, and yet

in the face of all this evidence of the hydrostatic inequilibrium of his injections, Ferber concludes : —

" The position of an exudation, taken all in all, is undoubtedly *chiefly* determined by the weight of the exudation, and by the position of the animal."

The weight of the fluid and the position of the animal are undoubtedly important factors in our problem, but one other factor is wanting to complete the solution, and that is, — the lifting and distributing force of the lung.

CHAPTER VI.

ANALOGY BETWEEN THE HUMAN LUNG AND THE DOG'S LUNG.

ONCE more I will state that for the present I am dealing only with normal lungs which have undergone no degeneration, and which are unimpaired in their action. Adhesions, parenchymatous modifications, and all other possible pathological complications of a case of simple serous effusion in the chest will be reserved for later consideration.

Like the dog's lung, the human lung is an exceedingly elastic body. Rokitansky says it is capable of contracting to one quarter, or even one eighth of its usual size. It follows, therefore, that the principles deduced from elastic bodies in enclosed spaces are as applicable to the human, as they were to the dog's, lung.

The human lung is always stretched beyond the zero point of its elasticity, and consequently there is the old struggle between elasticity and opposing forces. Increase the number of the antagonistic factors by the addition of a pleuritic effusion, and we have a condition of affairs very analogous to the dog's chest with its injected fluid. In both cases the fluid is introduced into the pleural cavity from parts without, and it matters not whether that introduction be brought about by action of the lung itself (see Note, Chapter IV.), by injection or by exudation, or by transudation, the statico-dynamical rela-

tions which are thereby established between fluid and lung must be the same in all cases.

The respiratory movements of a dog after a sudden injection of fluid into the cavity are usually somewhat violent for a time, but such movements cannot alter the physical principles which obtain between lung and injection. At best, they can only oblige a mutual readjustment of position.

We have seen that the rapidity of the injection, which was a matter of a few moments, could have no appreciable effect upon the adjustments between lung and fluid. No more can the rapidity of the exudation, which is a matter of days.

Compression of Lung. — So long as the effusion is suspended by the elasticity of the lung it certainly cannot compress the lung. On the contrary, we now see that it must exert a directly *negative pressure* or *traction*, and must therefore actually *retard* the retraction of that body. It will be impossible for the effusion to compress the lung until the latter has exhausted all of its elasticity, and become an inert, helpless mass of tissue. Just at what stage of an effusion this compression will begin I am unable to state. Rokitansky declares, however, that a normal lung is capable of contracting to one eighth of its usual volume. If such be the case, and the pulmonic tissue is normal, an effusion cannot be said to compress the lung until it occupies at least seven eighths of the thoracic cavity. Of course, if the lung is solidified to any degree, compression will be possible at an earlier stage.

We hear a great deal about the force necessary to drive a large exudation through the pleural membrane, and some entertain the idea that a portion of this force is stored up in the effusion, imparting to that fluid a sort of latent energy for subsequent development. This is

entirely a wrong idea. A certain amount of force is necessary to carry a drop of exudation through the pleural membrane in the same way that a certain amount of force, as expressed by a certain amount of coal, is necessary to carry an engine through a long mountain tunnel. As the engine emerges from the opposite end of the tunnel, however, both coal and its latent force are exhausted. So with the drop of exudation. When it emerges upon the inner surface of the pleural membrane, its impelling force is exhausted, and it becomes then passively subject to the new forces which grapple it. The force of exudation would of course come into play, if the chest were already so full of fluid that no drop more could enter without being compressed. Such a condition in the chest is hardly conceivable, however, and it will also be remembered that I am applying my argument now to those who talk of compression very early in the exudation stage.

Seat of the Exudation and Position of the Patient. — We saw that the point of injection and the position of the dog did not modify the principles involved, and the same is true of the seat of an exudation and the position of the patient. Suppose that a drop of exudation appears upon the inner surface of the pleural membrane, half way up the chest, and suppose, for a moment, that the retractility of that part of the lung in contact with the drop should hold the latter where it first appears. We have learned from our balloon that those parts most distended retract with the most force, and thus secure the water to the deprivation of weaker parts. From the application of this principle to the interpretation of our models, and from our knowledge of the anatomy of the lung, we now know that the lower part of the lung, being most distended, attracts to itself fluid to the deprivation of parts less energetic. Consequently our hypothetical drop will be drawn toward the comple-

mental space, and I believe that this must take place even against the action of gravitation. No doubt the force of gravitation is a powerful accessory factor in determining the distribution of an effusion in the chest, and its influence is especially conspicuous when an excess of effusion is present. So long, however, as the weight of a given amount of fluid is less than the difference between the attracting force of two parts of a lung, the fluid will accommodate itself to the stronger force in spite of the tendency of gravitation. I firmly believe, while an effusion is still so small that its entire weight does not exceed the difference between the elastic energy of the lower part and of other parts of the lung, that effusion will be held immovably in the complemental space, *i. e.*, between lung and diaphragm, and will be unable to gravitate away from that region whatever may be the inclination of the patient's body. The amount of water represented in the horizontal flask, on page 37, is as unable to gravitate when the flask is held upside down, as it is in the position indicated. The superior traction of the B C A segment of the balloon holds its sway supreme in every position you may place it.

The factors, with which we are dealing, however, are not fixed, but variable quantities, and hence the phenomena of adaptation must vary with every fluctuation in the balance of the antagonistic forces. When the stronger forces are satisfied, so to speak, then we behold the play of the weaker ones, and this, I believe, is the only true explanation of the peculiar phenomena which we have been studying.

The movements of the patient will exert some influence upon the adjustments of a very small effusion, since the jars, to which the thorax is thereby subjected, will either assist or retard, as the case may be, the progress of the fluid in the direction toward which it is most strongly at-

tracted. The movements of large effusions, however, would not be much disturbed by such an influence. The consistence of the effusion must also affect somewhat the rapidity of the adjustments, since a serous fluid flows more readily than a viscid one.

It is evident, therefore, whatever be the position of the patient, that an effusion is distributed in the chest, (1) according to the balance of retractility in different parts of the lung, and (2) according to the balance between the entire weight of the fluid and the lifting force of the lung, and an effusion can exert no great displacing influence until its weight exceeds that lifting force. Hence the bagging of the diaphragm, the obliteration of the intercostal depression, and other kindred symptoms can appear only in the order and manner already described.

I once more refer to the layer of fluid, which may be supported by capillary attraction, and, also, to the layer of fluid descending from the place of exudation, to say that they must be too insignificant in amount to appreciably affect the percussion sound.

As a general deduction from all these points of analogy between the human lung and the dog's lung, I conclude that the distribution of a pleuritic effusion must be somewhat as follows : —

With the patient in the perpendicular position, the effusion must collect in the complemental space. Thence it spreads upwards and forwards over the diaphragm, at the same time presenting a lateral surface or zone, which corresponds to the separation of the lower border of the lung from the line of the costal attachments of the diaphragm. The effusion and diaphragm are elevated by the retractility of the lung until the excess of the weight of the effusion begins to bag down the diaphragm and cause the intercostal depressions to be obliterated. As the

amount of the fluid increases, and probably *when it occupies about a third of the thoracic cavity*, it begins to be drawn up in the vertebral groove back of the lung, and, *lastly*, it comes between the lateral surface of lung and chest wall. Just when this last step in the process occurs I cannot say, but I believe the effusion must be very excessive before it occurs, and I shall show further reason for this belief later.

When the patient is in the horizontal position, the effusion must also collect in the complementary space and vertebral groove. Thence it extends up over the diaphragm and along the vertebral groove, forming the letter V, which I described in a previous chapter. Lastly it comes between the lateral surface of the lung and the chest wall. Every text-book says that an effusion begins immediately to press the lung away from the chest wall, but my models teach us, that whatever may be the position of the patient, the effusion appears *last* and in *least quantity* between the lung and the lateral chest wall.

Curve. — We have seen that when the dog was held perpendicularly and percussed, we obtained a letter S curve of flatness, which corresponded in *shape* to the lower border of the lung, and in *position* to the line of demarcation between the border of the lung and the injection.

We have also seen that the Ellis line of flatness is a letter S curve, which is very similar to the curve on the dog's chest, and which differs from the latter only to the extent that the curvature of the lower border of the human lung differs from that of the canine lung. The difference in the number of lobes between the dog's and man's lung cannot weaken our analogy, because, however many lobes a lung may have, it must have a lower lobe,

and that lower lobe must have a lower border, and that lower border is all that concerns us at present.

The natural deduction of all this reasoning is, that the Ellis curve of flatness corresponds in shape to the lower border of the lung, and in position to the line of demarcation between lung and effusion. As the exudation increases in amount, the curve of flatness rises, and, at the same time, tends to flatten out somewhat, until with excessive effusions, like Case III. of Dr. Ellis, page 8, it becomes nearly horizontal. The effusion must be very excessive, however, to make the line horizontal, for I have traced the curve as high as the third rib in the axillary line, and it still preserved its S form. During the absorption stage of the effusion the curve retraces the path passed over during the cumulative stage, and this is equally true when the effusion is rapidly withdrawn by thoracentesis as is represented in No. III. of Dr. Ellis' cases.

Remember that I do not claim that the S line of flatness must always appear under all circumstances, or that it will always present an invariable form. On the contrary I devote Chapter VIII. to a consideration of certain conditions which may modify the S curve or entirely prevent its formation. There are other conditions, also, which sometimes may prevent our tracing the line of demarcation between the lung and the effusion, even though the actual relations of the two bodies are the same as has been already described. (See Chapter IX.)

In order to illustrate how radically my theory, upon the formation and distribution of an effusion and upon the mutual relations between lung and fluid, differs from what is universally taught in the text-books, I will append a few quotations from leading authors: —

Gerhardt says, "A small amount of fluid, according to the law of gravity, gathers below and behind; as it

increases it allows the lung to contract. As soon as enough fluid has collected to cover the diaphragm pressure begins. The diaphragm is depressed, the heart is displaced, and the thoracic cavity is widened by lateral pressure. As the fluid increases it exerts a pressure *upward against* the lung; at first the lung swings upon the fluid and begins to be airless from below upward. That portion dips into the effusion and the lung is *pushed* up into the scapular region."

Da Costa says, "A moderate quantity of liquid only constricts the lung texture and leaves the bronchial tubes intact. A large accumulation compresses everything. It drives all the air out of the lung, pushes it into a small space against the vertebral column, and displaces the liver and heart." He also gives a diagram showing how a small effusion will round in the edges, and flatten the lower part of the lung. A second diagram shows a larger effusion separating the lung from the chest wall like a wedge with its base below.

Weil writes: "According as the exudation crowds in between the pulmonary and costal pleuræ, the lung retracts to a smaller volume. The diminished part of the organ still contains air, and swims upon the surface of the fluid which has the greater specific gravity. As the effusion increases, the adjacent portion of lung becomes airless from pressure, and dips into the fluid."

Guttmann says: "When absorption of a pleuritic exudation begins, the expansion of the lung increases, and the area of dulness diminishes. The intensity of the dulness also diminishes, since the thickness of the fluid — and thereby the distance between lung and thorax wall — is lessened."

I will not deny that the conditions described above may occur in extraordinary cases. I do assert, however, that such conditions are absolutely impossible in ordinary cases of pleurisy.

Hamernjk presents still another and original theory of compression. He believes that the simple weight of a fluid is in no wise capable of expelling the air from a portion of lung, and that it is just as little able to expand the thorax, or displace the mediastinum and diaphragm. He thinks that these changes are brought about by means of forced expiratory movements, and that they appear when the pressure exerted upon the thorax wall during expiration can be transmitted to the above named organs by means of the pleuritic exudation.

Fraentzel and Jaccoud both recognize that the lung is elastic and that it will retract before the exudation, and they say that it cannot be compressed until it has reached the neutral point of its elasticity; and yet later Fraentzel repeatedly refers to the resistance of the lung to the fluid in endeavoring to explain certain of the symptoms of percussion. Neither of these men, apparently, has the remotest idea that the lung lifts the fluid as it contracts, while this very idea forms the *pith* of my essay.

CHAPTER VII.

ON THEORIES.

Most of the authors whom I have consulted seem to consider the pleuritic curve of flatness as a very uncertain sign, and of little importance when found, and therefore they pass it lightly over. A very few of them have ventured an opinion as to its origin, and a short review of these opinions will be found entertaining.

Paul Niemeyer says: "The exudation collects first in the lower and posterior part of the thorax, and when this part is full, it extends also forward, whence it is evident that its niveau is higher behind than in front."

I never saw it asserted before that the shape of the bottom of a vessel has any influence upon the level of the surface of any fluid which it may contain.

Gerhardt says: "The line of exudation is not a perfectly straight line, but, as Damoiseau has shown, on the side of the thorax it presents numerous undulations. I find no other ground for these undulations in the curve than the irregularities in the thickness of the breast wall, which are due to the insertion of muscles."

The slightest familiarity with the true S curve of flatness will show the entire inadequacy of this explanation.

Peter is more enthusiastic, and enters into the subject with considerable vigor. According to him, the "curve is that which one obtains on passing a secant plane obliquely through the cavity of the chest." He says that

the position of the patient, the form of the thorax, the presence of exudation in the pleural cavity, the nature of the exudation, and, finally, the action of gravity, are the elements for the solution of the problem.

I. The Position of the Patient. — If the patient lies horizontally, the fluid obeys the law of gravity and collects in the vertebral groove. When the patient is lying at an elevated angle, the fluid sinks to the lower part of the groove.

II. Peter claims that the nature of the exuded fluid is of great importance, and says that this point was neglected by Damoiseau.

If the exudation be entirely serous, the line is nearly horizontal.

If the exudation be entirely fibrinous, the exudation will adapt itself slowly to changes in the position of the patient, and the line of flatness will be more parabolic in shape.

If the exudation be sero-fibrinous, there will be two zones of dulness.

a. A superior zone of superficial dulness, due to the thick fibrinous exudation which cleaves to the wall of the chest.

b. An inferior zone of profound and absolute dulness caused by the serous exudation which gravitates downward.

The upper border of these two zones combined will be limited by a line, which is curved in the superior part (matité de la matière fibrineuse), but the line becomes horizontal as it is prolonged towards the sides and below (matité de la serosité).

To illustrate his meaning Peter partially filled a bottle with tar water and tipped it to an angle with the floor. Replacing the bottle horizontally again, he obtained the curves represented in the following figures.

The region A B (Fig. 19), which is still bathed with the viscid fluid, corresponds to the dull region of the fibrinous matter in the chest. Percussion of the parabolique couch, *i. e.*, of the zone A B, gives only a relative dulness. The dulness is absolute, however, in all the horizontal couch B C.

This theory of Peter is very ingenious, and applies very well to the phenomena of his bottle, where the tar water assumed a hydrostatic level because nothing prevented it from doing so.

My bottle experiments, however, prove that an exudation in the chest differs from the tar water in the bottle

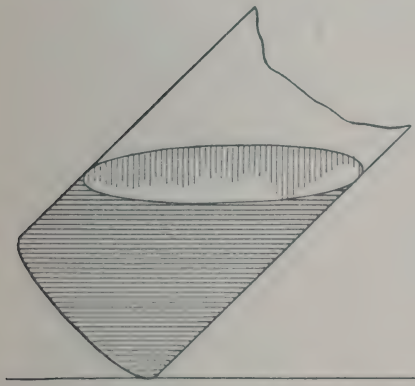


Fig. 18

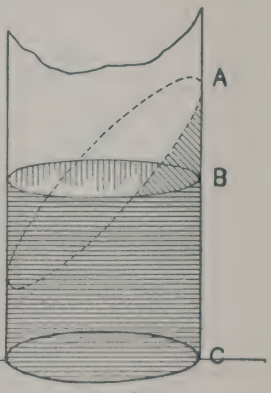


Fig. 19.

in that it cannot assume a hydrostatic level, because the elasticity of the lungs prevents it from doing so. Examination of Model III. shows that the fluid must be drawn up the entire length of the vertebral groove when the patient with a large effusion is reclining at an angle to the bed, and hence no such line of demarcation could be obtained by percussion on the back of the chest, as is visible in the bottle.

Fraentzel reiterates the theory of Peter, and says that the fluid assumes a level in accordance with its specific gravity, and that the curve is higher behind, and thence descends to the sternum with its concavity directed forward, when the patient reclines in an elevated position.

That such is an approximate, but not an accurate, statement of the case, is evident again from Models III. and IV. I think it would be very difficult for any one to trace on the back a true line of demarcation between dulness and flatness, under the conditions represented in Model III. On the other hand, the position of the curve M B, in Model IV., would no doubt remain as distinct in the horizontal position as when the dog was in the vertical position, and we have clinical evidence that a portion of the curve does persist in front even when a patient lies upon his back.

Fraentzel was unwittingly treading upon the very verge of the discovery of the true relation between lung and effusion when he wrote: "So long as the lung, which under normal conditions is always expanded, is able to retract before the exudation by virtue of its contractility, it still contains air, and it can suffer no compression from the exudation until it has reached its point of equilibrium." This is all true, and it lacks but one thing more to make it complete. Fraentzel failed to perceive that the lung, by virtue of the strength of its contractility, takes the effusion along with it in its retraction, and that thereby the latter assumes a pneumono-dynamic, instead of a hydrostatic, level.

Damoiseau performed the following experiment, in search of an explanation of his curve. He introduced a canula into the trachea of a cadaver, and inflated the lungs until the intercostal spaces protruded. Then, on removing the external parts of the thorax, he found the

lung everywhere in apposition with the chest walls. Then he allowed the air to escape slowly, and watched the effect. The parietal and visceral layers of the pleura commenced to separate from each other, and this separation began first at the lowest point of the costo-diaphragmatic groove. Then little by little it spread front and back toward the anterior and posterior median lines. The lower border of the lung became almost horizontal, and the diaphragmatic pleura below was in immediate contact with the ribs. With continued aspiration one soon saw a kind of parabola form below the inferior angle of the shoulder-blade. Its summit rose, its borders separated, and it underwent the same changes in an inverse order which the curve presents during the absorbent stage of a pleuritic effusion.

He infers, therefore, that the lungs must act similarly in case of an effusion and that the fluid by *virtue of its gravity* must be situated in the lower portion of the costo-diaphragmatic groove and in the hypochondriac and axillary regions.

"Let us suppose, now," he says, "that the serum interposes itself between the layers of the pleura with or without cystic pseudo-membranes, what reason is there that the pulmonary pleura does not act with the liquid as with the atmospheric air? I can only see the superior weight of the liquid which can change the conditions. But that weight which is powerless to modify the results in the horizontal plane, will act in concourse with the concentric elasticity of the lung in the vertical plane, since that part of the pleura which is lowest, is also the most excentric, and we know that the concentric elasticity of the lungs is, in general, proportional to the length of the bronchial tubes."

In reply to Damoiseau's query I will say that the lungs will act in precisely the same manner with a given

volume of effusion as with the same volume of air. The difference in the results will be due simply to the difference in the negative pressure, *i. e.*, in the weight, of the two fluids acted upon. The volume of air exerts practically no negative pressure, whereas, we have seen, in Experiment II., Chapter III., that the weight of the effusion must modify to some extent the shape of the lung.

In the second part of the quotation Damoiseau says, that the weight of the fluid "though powerless to modify the results in the horizontal plane will yet act in concert with the concentric elasticity of the lung in the perpendicular plane." For my part, I cannot conceive how the weight of a fluid can act directly *upward*. Owing to its incompressibility a fluid may displace bodies upward, if those bodies come within the influence of that displacing force. But the relation between a lung and an effusion is exactly the reverse of that implied in Damoiseau's theory, and hence that theory must be laid aside.

Felix Niemeyer says: "The dulness proceeding from pleuritic effusions generally first becomes perceptible in the region of the back and below the scapulæ. As it ascends it spreads toward the front. The dulness scarcely ever extends as far upward in front as it does behind. . . . . Posteriorly, as the upper limit of the effusion is approached, the dulness gradually becomes fainter and less distinct. The reason for this is, that the thickness of the body of effusion upon which the dull sound depends gradually diminishes from below upward."

Ferber says, that the position of the exudation depends upon two conditions, namely, the specific gravity of the fluid and the position of the patient. Given these two conditions, the level of the fluid will always be horizontal.

He says, however, that the exudation is in a condition

of constant motion in the chest and that the ensemble of these movements constantly causes the border line of the exudation to oscillate upward and downward. This condition he deems important in the production of the curve formed line of Damoiseau, for he thinks that the lungs, as they are moved up and down by the oscillations of the fluid, become caught by adhesions here and there, and hence the undulations in the otherwise horizontal line of flatness. He says, further, "If the exudation lies upon the diaphragm the impression upon the model does certainly rise from behind forwards. But this is not to be confused with the line of dulness of the exudation, as it appears on the thorax, for the latter corresponds to the superior border line of the layer of exudation, sometimes thinner, sometimes thicker, which lies between the lateral surface of the lung and the thorax wall. If Garland did not find such conditions in certain cases, it was probably due in part to the rapid solidification of the material employed by him, and in part to the fact that some of his experiments were performed upon dead animals."

Ferber entirely ignores the fact that I carefully percussed the dog before opening the chest, and that having marked the line of flatness I found that it accurately corresponded in shape and position to the lower border of the lung, and *not* to the upper border of the occasional, and *very thin* layer of fluid between lung and chest wall.

My own theory in regard to the letter S curve of flatness is this. The curve depends upon: —

1. Presence of fluid in the pleural cavity.
2. Position of the patient.
3. Elasticity of the lung.
4. Shape of the lung.
5. Negative pressure of the fluid.
6. Shape of the chest.
7. Absence of complications, adhesions, etc.

The curve appears in its purity only in the vertical position of the body. The lung lifts and distributes the effusion according to the balance of its elasticity in different parts, and the shape of the lung is modified somewhat by the negative pressure of the fluid. The natural convexity of the chest of course bends the whole curve correspondingly, but otherwise the shape of the chest has nothing to do with the curve. It is the same for all chests, and on a dog's chest it differs only as the lower border of the canine lung differs from that of the human lung.

CHAPTER VIII.

CONDITIONS WHICH MAY MODIFY THE CURVE IN PLEURISY.

It will be remembered that my argument thus far has been, for the sake of simplicity, limited to cases which are uncomplicated by adhesions or by pathological changes in the lung itself.

I will now consider certain complications incidental to the patient and to the disease, which may modify the curve of flatness. First and foremost among such causes are *adhesions*.

Of course I exclude from consideration all cases of small so called circumscribed pleurisy, because such cases may assume any conceivable form and position.

Whenever adhesions occur, they will compromise the action of the lung, and just so far as they hamper the free play of that body they must modify the line of flatness. Some have gone so far as to maintain that this curve is the result of adhesions. No theory could be more diametrically opposed to the real truth of the case. We have seen that the curve of flatness has a constant letter S character, and hence the cause which produces it must be invariable, and it is very improbable that adhesions are formed with sufficient regularity to account for so constant a phenomenon. Moreover, we have seen that in the experiments upon the dogs, where pleural adhesions were always wanting, the curve was always the same when the dog was suspended vertically. Dr. Ellis assures me

that in all his cases the curve has been best marked and most definite in character during the early stages of the effusion, when firm adhesions at least had not had time to form. The curve persists during absorption, but is often more difficult to trace in that stage. We see, therefore, that those conditions which are most unfavorable to adhesions are most favorable to the formation and detection of our curve. Moreover, I do not believe that adhesions are so common in ordinary acute pleurisy as some suppose. Mohr says in his statistics upon pleurisy that twenty-three cases out of forty-nine were free of adhesions, that is to say, adhesions were wanting in forty-seven per cent. of the cases analyzed by him.

Change of Posture. — We have seen in our models that change of posture with injection of any considerable amount does effect a change in the position of the fluid, and hence it must modify the line of flatness. This modification, however, is chiefly visible in the posterior part of the chest, while the line of demarcation between lung and fluid is but little altered in front. (See Model IV., page 29.)

Permanent retention of one position can modify the curve only as it favors the formation of adhesions corresponding to the position assumed. As soon as the patient resumes a perpendicular position, relations essential to the curve must establish themselves if adhesions are wanting, and hence change of posture can in itself affect the curve only temporarily. I imagine that the adjustments between the lung and the effusion for different positions of the body will be slower when the pleural layers are clothed with a very viscid secretion than when they are bathed with simple serum.

Diminished Contractility of Lung. — Any modification of the lung parenchyma which diminishes the elasticity of that tissue, must of course affect the relation

between lung and effusion, and ought, consequently, to modify the curve. Thus, if a lung is solidified by catarrhal or tuberculous deposits it loses in elasticity and gradually becomes a solid resisting mass which would be liable to compression by the effusion much earlier than under ordinary circumstances. It is a remarkable fact, however, that the lung may be solidified to an excessive degree, as indicated by the percussion sounds on both sides of the chest, and yet the curve of flatness may still be clearly made out by careful, light percussion. I can conceive also, that the pleuritic curve would be abnormal if the elasticity of the lung were impaired by excessive and prolonged distention of emphysema.

We have seen that the rapidity and seat of the exudation can have no effect upon the curve, and an analysis of Dr. Ellis's cases shows that the nature of the exudation does not alter the curve. In Case III. eighty-four ounces of clear, yellow serum were drawn off. In Case V., service of Dr. Minot, twenty-four ounces of pus had been removed three weeks previous to the patient's entrance into the hospital, and aspiration revealed pus still present. A comparison of the curves in these two cases shows absolutely no difference in their general features, and it must be remembered also, that they were drawn independently by different persons. The curve undergoes some modification as the effusion increases in size, but the exudation must be enormous before its S feature is obliterated.

I have frequently referred to a thin, narrow layer of fluid, which is drawn in between the lower edge of the lung and the chest wall with medium injections. Ferber noticed the same thing and places great emphasis upon it. Indeed, it is the basis of his theory of the line of dulness.

If, however, the lung is removed from the chest wall

only by a "very thin" layer of fluid, as Ferber says himself, it cannot certainly be said to be very much collapsed, and hence its resonance will overpower the flatness of the fluid. We know how difficult it often is to detect solidified lobules of considerable size, owing to the resonance of the surrounding tissue, and therefore I think it is rather stretching the point to lay so much emphasis upon the intervention of a "very thin" layer of fluid. But even allowing the utmost importance to this thin layer, it would only be able to cause a slight dulness. Dulness, however, is not flatness, as I cannot too often repeat, and, therefore, the line of demarcation between dulness and flatness would remain unaffected.

The respiratory movements, when violent, probably may affect the curve somewhat. I cannot believe, however, that such combined movements of lung, fluid, and diaphragm, can materially alter the mutual relations of these three factors. The lung and effusion are bound together in a particular relationship, and therefore I should expect that they would move up and down during respiration as one body unless their mutual union were broken by sudden shocks and jars. This point, however, will be again referred to, and will be more fully treated on a subsequent page.

CHAPTER IX.

CONDITIONS WHICH MAY RENDER THE CURVE DIFFICULT TO TRACE.

When a lung retracts before a pleuritic effusion it diminishes in resonance; and the duller that resonance becomes, the more difficult will it be to distinguish the same from the effusion flatness below. All changes in the lung parenchyma which tend to diminish the pulmonary resonance also tend to increase the difficulty of tracing the curved line of flatness.

Solidification of the lung, hypostatic congestion from persistent retention of one position, saturation of the lung parenchyma with inflammatory products, thick fibrinous deposits upon the pleural membrane, — all such conditions are causes of perplexity. It is astonishing, however, to notice the delicacy with which careful, light percussion will still bring out the distinction between pulmonary dulness and effusion flatness, even when the former is so intense by reason of solidification of the lung that it appears flat itself by comparison with the resonance of the neighboring lung. With reference to this same point Anstie says: "Over the whole space occupied by the fluid there is found a dulness more pronounced in some cases than in others, but always with a character of its own which must be heard to be recognized, but which is much more marked than that produced by lung solidification."

Œdema of the lung is a most culpable cause of con-

fusion, because the presence of a fluid in the alveoli excludes a part of the air and thereby renders the sound of the lung almost as flat as that of the pleural transudation. The œdema in the lung tissue gravitates to the lowest points of support, and hence collects most abundantly in the posterior inferior part of the lower lobe. This part of the lung, however, is the part included within the limits of the dull triangle which I have already described. This triangle is always the dullest portion of the chest when fluid is present in the pleural cavity ; and if this dulness be increased by saturating the lung with œdema, the percussion sound may ultimately be converted into flatness.

If we draw the base-line of our dull triangle from the summit of the curve perpendicularly to the vertebral column, and if we imagine the lung beneath to be saturated with serum so that its percussion sound is very dull, we shall readily understand why the books are so unanimous in declaring that the line of dulness with hydrothorax is horizontal. The dulness of the dull triangle, however, is not always so intense as I have here supposed it, and I believe that its percussion sound can almost always be distinguished from the flatness of the neighboring transudation, if proper pains be taken in percussing. I have observed a case of bilateral hydrothorax from cardiac lesion where I traced a curve on both sides, and where I had no difficulty in satisfying others as well as myself of this fact. I am inclined to think, therefore, that in most cases of hydrothorax, where the line of dulness has been declared to be horizontal, a more delicate percussion would have demonstrated the S curve of flatness as usual. In tracing the curved line of flatness, under all circumstances, the importance of recognizing the dull triangle cannot be over-estimated, because the portion of lung which corresponds to this triangle is the portion which

gives the characteristic S shape to the lower part of the curve. It is, however, the most dependent portion of the lung, and is therefore the most liable to many accidental casualties which will modify its resonance. Thus the dulness of hypostatic congestion is most evident within this triangle. The muscles of the back are exceedingly thin over it. It is least ventilated during quiet, superficial respiration, and, therefore, if the person percussing does not give especial attention to the triangle, he will fail to distinguish that its percussion sound is dull and not flat.

The rule which I gave in the first chapter, to let the well side of the chest alone when endeavoring to trace the curve of flatness, is especially applicable to percussion over the dull triangle.

The best way to distinguish between the dulness of the triangle and the flatness of the effusion is to percuss out laterally from the vertebral column in short lines, and very lightly.

It often happens that a patient has been lying down or sitting quietly for some time previous to the examination, and in consequence of this the percussion sound upon the back, and especially within the dull triangle, is found to be very dull.

I have seen pleuritic patients with consolidated lungs on whom it was almost impossible to trace the curve after they had been sitting quietly for some time. Let such a patient take a few deep inspirations, or, if possible, let him walk a short distance in the open air, and when he returns the distinction between dulness and flatness will be as clear as that between a shadow and its penumbra. I happened one day to percuss a pregnant woman who was about five or six months along, and who had a slight bronchitis. I found the region corresponding to the dull triangle so very dull that I at first suspected pleurisy.

Yet, in percussing out from the vertebral column I could find no flatness until I reached the normal flatness of the liver. Accordingly I made the woman inspire deeply a few times, and the dulness diappeared. It immediately occurred to me that a pregnant uterus at full term might push up the abdominal organs so high that the flatness of the liver might be mistaken for pleurisy, with its curved line, dull triangle, and all. Accordingly I obtained permission to percuss a large number of pregnant women who were near their full term, in Professor Braun's wards in Vienna, and I thus convinced myself that the upward displacement of the liver was not sufficient to lead any one astray who is at all skillful in percussing.

This brings me to the consideration of certain difficulties of percussion which are more subjective than objective. I believe that the curve of flatness often escapes detection, because the person who is percussing neglects to observe a simple rule, which is, *always percuss in straight lines.* I have seen so many fail to define the outline of some internal organ, like the heart, for instance, and simply because they percussed round and round hap-hazard, without any idea of any direction, that I have laid it down as the golden rule of percussion : —

Always percuss in straight lines, and pursue each line to its terminus before taking up another.

I have already described in Chapter I. my manner of percussing, and I will only renew the advice to percuss lightly.

In speaking of the absolute dulness on the parts of the chest from which the lung has been entirely removed by the effusion, Fraentzel says, " The percussion, moreover, must never be too strong, else it may easily become impossible to perceive the absolute dulness of the sound ; for, with very strong percussion either the well lung, or the ribs on the affected side are set in vibration, and the

sound no longer appears absolutely dull. Many times physicians have disputed with me regarding the presence of absolute dulness, simply because they have percussed too strongly, and lighter percussion has persuaded them of their error." This advice is particularly applicable to the tracing of the line of flatness.

The one mistake *par excellence* in percussion of the chest is the neglect to distinguish between dulness and flatness. The German literature is very indefinite regarding the distinction between these two terms, at least so far as their application to pleurisy is concerned. I cease to wonder that the Germans find so many curves of dulness, and so many modifications of the same, when I see the reckless manner in which they interchange the terms dulness and flatness. For instance, Oppolzer talks about the percussion sound being *dumpf* (dull) and *leer* (flat) from below upward, but he evidently considers it of no importance to designate where the sound is *leer* and where it is *dumpf*. Again, Felix Niemeyer employs the word *leer* with an evidently different signification from that which Oppolzer gives it. He says, "the percussion sound is (gedämpft) dull over the fluid. Over those parts where the lung lies against the chest wall in a state of contraction, though still containing air, the percussion sound is *leer* and tympanitic."

Now a sound cannot at the same time be flat and tympanitic, hence Niemeyer must have had some other meaning in his mind. I see in the American edition of Niemeyer's work the editors have translated the clause "*hollow* and tympanitic." A tympanitic sound, however, is a hollow sound, and, therefore, the words of the translation are synonyms. I think it is more probable that Niemeyer meant "dull and tympanitic." Gerhardt speaks of the Dämpfung (dulness) increasing in intensity from above downward.

Skoda defines the word *leer* in a twofold manner, somewhat difficult to understand. In the first place he contrasts a *leer* sound with a *voll* one, and says: "If one percusses on different parts of the thorax or abdomen with equal force, he will find that the sound is more persistent and apparently extended over more area on some spots than on others. The first kind of sound I call the *voll* percussion sound, and the second I term less *voll* or *leer*." Explaining his meaning still further, Skoda says the difference between *voll* and *leer* is independent of the pitch, force, or duration, of a sound. I think Skoda's meaning is this: Every musical instrument gives forth a compound tone which is composed of a fundamental sound with its superimposed harmonics. When the secondary or accessory waves of sound flow harmoniously with the fundamental waves they automatically magnify the effect of the latter and we obtain a full, rich tone. When, however, the accessory waves alternate, as it were, with the fundamental ones, we obtain an imperfect sound. This difference in quality, as Skoda correctly says, is independent of the pitch, duration, or initial force of a tone. Moreover, it does not depend upon the kind of instrument employed but upon the condition of that instrument, and hence the difference between a good and a poor instrument, or between the tones of the same instrument under different conditions of dryness and moisture, heat and cold. Every one familiar with the flute is aware of the change in the fulness of the tones of that instrument as it becomes warm with the breath. I think, therefore, that Skoda wishes to express some such difference in the quality of the percussion sound when he distinguishes between *voll* and *leer*.

In another place, however, he employs the word *leer* as synonymous for *flatness*, and thereby indicates an absence of resonance. He says, "a perfectly *leer* sound, such as

one obtains upon the thigh, shows that the space beneath the point percussed contains no air or gas, but is occupied by fluid or airless tissues." I submit that this application of one word to two entirely different conditions must inevitably cause confusion and lead to misunderstanding and I would therefore respectfully suggest that the word *leer* be more restricted in its application or that its use be discontinued.

The German writers are not the only ones guilty of this careless confusion of terms. The error is universal. Even Flint, who is ordinarily so clear in distinguishing between dulness and flatness, loses sight of that distinction sometimes. He speaks of "the line of demarcation between the dulness *or* flatness and pulmonary resonance," as if dulness and flatness were synonyms as applied to the percussion sound over an exudation.

I have already defined in Chapter I. the distinction between dulness and flatness. Ordinarily, in pleurisy the difference in the two sounds may be sharply defined by correct percussion when any considerable amount of effusion is present. I wish, however, to assure those who read this book that they will always fail to detect the S curve of flatness until they learn to distinguish between flatness and dulness.

CHAPTER X.

DIAGNOSTIC IMPORTANCE OF THE ELLIS CURVE.

We have seen that the letter S curve is a constant factor when free fluid is present in the pleural cavity. We have proved that it corresponds in shape to the lower border of the lung, and indicates the line of demarcation between lung and fluid. No other pathological conditions within the chest are capable of producing a similar curve, and hence it must be considered as pathognomonic of fluid in the pleural cavity. The curve is in no wise indicative of the nature of the fluid, as is conclusively proven by my experiments and by Dr. Ellis's cases. I have shown, moreover, that those conditions which are most favorable to a typical curve are least favorable to adhesions; hence a well-marked curve, retaining its true characteristics throughout the ebb and flow of the exudation, will indicate the probable absence of adhesions. The reverse of this statement is equally true.

Tanner says: "I do not believe that any amount of cold by itself will produce the disease (pleurisy) in a healthy individual. It may prove the exciting, but not the essential, cause of the inflammation. The statement has been made that in the greater number of cases of pleurisy on the right side, the inflammation depends on the preëxistence of tubercles in the lung, while pleurisy on the left side is usually independent of this cause." Prof. Schrötter, of Vienna, entertains similar opinions

in regard to the association of pleurisy and pulmonary diseases.

I think, however, a well marked contrast between the dulness of the dull triangle and the flatness of the effusion indicates that the lower part of the lung at least is normal. On the other hand, if the curve is difficult to trace with a small pleuritic effusion, owing to an excessive dulness of the pulmonary resonance, one should be suspicious of trouble in the lung itself. The same difficulty in tracing the line with hydrothorax would point simply to œdema of the lung. I believe that a more accurate knowledge of the true curve of flatness will demonstrate that the size of pleuritic exudations has been hitherto, as a rule, greatly overestimated. The neglect to distinguish between pulmonary dulness and effusion flatness must frequently have led to exaggerated statements of the amount of fluid in the chest. If one recognizes only *one* percussion sound of varying intensity from base to summit, he exclaims, "An enormous effusion!" Had he percussed more carefully, and thus detected the line of flatness with dull resonance above it, he would have said simply, "A very large effusion!" Ferber says: "It is only rarely that one finds an absolute dulness as high as the apex."

My own experience with regard to this point coincides with that of Ferber, and therefore I think that an effusion which is sufficient to completely fill the chest and to compress all air out of the contracted lung, is one of the rarest of pathological conditions.

The testimony of pathological anatomists upon this point is valueless, because they usually announce merely the number of pints of effusion found in the chest. The measure of the amount of the fluid, however, is of value only with reference to the capacity of the chest. An enormous effusion for some persons would only be moderate for others.

Peter boasts that he has rescued the curve from the opprobrium of being merely a physical curiosity and bestowed upon it an abiding honor by making it distinctive of the nature of the exudation. "Hence," he says, "the curve is diagnostic (almost geometrically) of the nature of the exudation," and he bases his prognosis and treatment upon this hypothetical virtue of the curve. My experiments prove conclusively the entire fallacy of such a theory. Be the exudation what it may, so long as it is fluid, the line of flatness for the perpendicular position will be the letter S curve, if the lung tissue is normal. The upper curve of dulness which Peter describes upon the bottle may or may not persist after the bottle is set up erect, but I have shown that the movements of free water in a bottle cannot be compared with the movements of a fluid which is in a state of suspension.

CHAPTER XI.

INTERPRETATION OF VARIOUS PHYSICAL PHENOMENA OF PLEURISY.

WHENEVER in the process of scientific thought a new theory is promulgated it is always received with suspicious reserve. However logical may be the arguments in its defense, and however accurate may seem the experiments in illustration of it, the evidence must all be corroborated by other observers, and the theory must be submitted to the crucial test of a practical application to observed phenomena.

If by such an ordeal it is shown that the new theory can better interpret a larger number of facts than any other theory previously advanced, then it is accepted and is accorded such honor as is due to its importance.

I began this essay with the avowed purpose of seeking the interpretation of the writing on the wall, — the curved line of flatness. In the process of that search I have discovered a relationship existing between a lung and a pleuritic exudation, which has never been described by any other author. I do not pretend that I have just discovered the fact that pulmonary tissue is elastic. Everybody knew that before. I do claim, however, that I have overthrown the hypothesis, hitherto universally accepted, that the lung is compressed and driven away from the chest wall by an encroaching exudation, and I have substituted for that hypothesis the assertion that a

fluid exudation exerts a negative pressure upon the lung by virtue of its weight.

More than this, I have demonstrated more clearly than any of my predecessors the importance of the pulmonary elasticity, in that I have shown that it is capable of supporting the *total weight* of a large body of effusion.

I propose now to analyze various other physical signs of pleurisy, to show wherein the theory of fluid compression as hitherto advanced is wholly inadequate to explain such signs, and finally, to prove that my own theory of the mutual statico-dynamical relations between a pleuritic effusion and a lung affords the only true explanation of those signs.

I will therefore take up in succession certain of the physical signs of pleurisy, and analyze them according to our new light.

Flatness is the sound derived from non-resonant bodies. It may always be obtained on that part of the chest where lung has been replaced by any considerable amount of effusion, provided the percussion be properly made. By "properly made," I mean that the percussion should always be light. If the percussion be applied over the contracted lung, the sound will be dull, not flat.

Dulness. — Any condition which diminishes the resonance of the lung causes dulness. In cases of pleurisy the lung above the effusion is dull. The dulness is usually least marked at the apex, and thence increases in intensity as the percussion approaches the line of flatness. It is always greater behind than in front, and appears to reach a higher level there (Wintrich's curve).

All the authors whom I have consulted say that this dulness is chiefly due to the interposition of fluid between lung and chest wall, and some embody this idea in diagrams. It would take too long to describe the variations which have been played upon this theme. It seems to

me very strange, however, that among so many possible causes of dulness one should give such prominence to a purely hypothetical cause. In Chapters VIII. and IX. I have enumerated a number of conditions which are common complications of pleurisy, and any one of which is sufficient to produce great dulness. The fact that the chest is partially filled with fluid, and that the pulmonary capacity for air is thereby diminished, is reason in itself for dulness. Moreover, on every normal chest the percussion sound on the back is duller than it is in front, owing to the thicker muscles of the back. Yet men grasp at the one idea: "Dulness, ergo effusion between lung and chest wall," and ignore all other more probable causes.

First Appearance of Dulness, and of Flatness. — The analysis of my models has shown that the effusion collects first in the lower posterior portion of the chest, and thence spreads up over the diaphragm. A narrow zone appears along the costo-diaphragmatic groove, and as the edge of the lung above retracts this zone increases in height. (See Models I. and II.) It is obvious, therefore, that the flatness of the effusion, and the dulness of the contracted lung, must both be first evident in the lower posterior part of the chest. I doubt, however, if the flatness of the fluid can ever be distinguished at the very beginning of affairs. The zone of fluid is so narrow that its flatness, like that of a small solidified lobule, will be obscured by the associated resonance of the lung. The combined effect, therefore, ought to be a general dulling of the percussion sound along the lower posterior border of the lung, and German authors are unanimous in the testimony that such is the actual case.

The conditions of percussion in the axillary region are very different from those upon the back, owing to the relative thinness of the walls in the former region. Nice distinctions in the percussion sound, therefore, can be made

out more delicately and more easily in the axillary region than elsewhere on the chest. Under all circumstances it is easiest to distinguish flatness from dulness in the axillary line, and consequently as the small zone of effusion is spreading across the chest, its flat sound, as compared with the pulmonary dull sound, becomes first distinguishable in the axillary region. Dr. Ellis tells me that in the early stages of suspected pleurisy he has always been able to trace the curve of flatness first in the axillary region. This is not because the effusion collects first in that region, but the conditions of percussion on the side are more favorable than on the back, and therefore the line of demarcation between lung and effusion can be distinguished first in that region. It is evident, therefore, that the parabolic curves of Damoiseau, with their branches gradually spreading out in either direction to sternum and vertebræ, are only ideal figures which correspond to no actual conditions within the chest. The curve is not a parabola, but a letter S, and that portion of the curve which appears first in the axillary region is not the section of a parabola, but a short section of the letter S.

Tympanitic Resonance. — Very often the pulmonary resonance in pleurisy presents a tympanitic character, and this is explained by the diminished tension of the lung. I imagine that this explanation is correct, but I object to the accompanying assertion that the tension of the lung is diminished by the compression of the effusion. Fraentzel describes the conditions under which tympanitic resonance sometimes occurs, as follows: "If the pressure to which the lung has yielded is not very great, we sometimes observe that the sound in the upper part of the affected side of the chest is again heard somewhat louder, high-pitched, and tympanitic; then again, after a longer or shorter time, it becomes absolutely dull.

This symptom, at the first moment most striking, we observe after violent fits of coughing, and therefore, especially, after raising such patient for the purpose of examination, a process which often brings on violent paroxysms of cough. The obvious conclusion in this case is, that the lung, being compressed by a slight and therefore easily overcome pressure, becomes again partially dilated by the violent fits of coughing; if the fit of coughing cease, then also the counteracting pressure against that of the effusion gradually disappears, the lung is again compressed, and the sound becomes the same as before the fit of coughing."

The phenomena described in this quotation were first observed by Traube, and Fraentzel has adopted Traube's explanation of the same, without any apparent misgivings. It appears to me, however, that the explanation is entirely wrong. The first and all-essential condition of the phenomenon is that the lung shall be in a state of expansion and shall contain air; and this condition, as I have shown, is incompatible with compression by the fluid. I should explain the phenomenon as follows: A fit of coughing is simply an explosive form of respiration, and is accomplished by a violent action of the respiratory muscles. The inspiration is abnormally full and deep, while the expiration is impulsive and vigorous. Hence it is obvious that the increased expansion of the lung and the increased amount of air therein are due simply to the magnified excursions of the chest wall. When the irritation which produced the cough subsides, the respiratory efforts become weaker, the chest wall collapses, the lung contracts again, the volume of air within the lung diminishes, and the tympanitic resonance vanishes with the restoration of the original dulness. It is evident that a certain amount of pulmonary expansion is essential to the production of the tympanitic resonance,

since that sign is absent when the patient lies upon his back breathing superficially, and it appears only when a certain degree of lung tension is established by the fit of coughing. Hence I do not believe that this symptom could ever occur if the effusion were of sufficient size to actually compress the lung.

Change of the Level of an Effusion corresponding to Changes in the Position of the Patient. — This is a much disputed point, and has been earnestly contested by many authors. Skoda says, "The assertion that the dull percussion sound with a pleuritic exudation changes its place with the various changes in the position of the patient, is wrong in the majority of cases." The dulness remains stationary, and he explains the fact by asserting that the lungs almost always become adherent to, or grow to, neighboring parts in the region of the exudation, and hence the fluid is maintained in one position. Wintrich also believes in the early encysting of an effusion and a resulting immobility.

On the other hand, Fraentzel, Ferber, and Weil, unite in declaring emphatically, that the upper border of an effusion does change its level with changes in the position of the patient. Now all these authors are partly right and partly wrong in their statements. Some effusions, certainly, do change their level on change of position, as indicated by percussion, and some do not, and I think I have clearly laid down the principles which will explain this seeming paradox, and remove the confusion in regard to it. On page 63, I said that a small effusion which is completely in the power of the lower part of the lung cannot change its position, whatever be the inclination of the patient's body. With large effusions, however, there is an excess of fluid, and this excess must inevitably flow hither and thither, according to the movements of the patient. Moreover, there must be a certain combined

movement between the lung and the fluid, analogous to the adjustments presented in our horizontal flask on page 37. We there see the curve of the balloon more convex at B, and flattened a little at A.

I am delighted to find clinical evidence of the correctness of my theory regarding this point in Fraentzel's article on pleurisy in Ziemssen's "Handbuch." Professor Fraentzel there says if the line of dulness of an effusion, which extends as high as the third rib in front, be marked upon the chest in the erect position, and be again marked with the patient lying down, it will be found that the level of the fluid has descended an inch in the latter position. That is to say, as the patient lies down the anterior part of the lung and the effusion mutually descend in front as does the balloon at point B, and it must be equally true that the posterior part of the lung will be affected in a manner analogous to the balloon at A, although we may not be able to trace that change by means of percussion.

Fraentzel and Weil are correct, therefore, in regard to large effusions, while Skoda and Wintrich are equally correct, so far as small effusions which are associated with energetic lungs are concerned. The last two err, however, in the assumption that adhesions are essential to the immobility of the fluid.

Changes in the Level of an Effusion, corresponding to the Respiratory Movements. — Gerhardt mentions certain cases of pleurisy where he was able to detect by percussion slight oscillations in the upper boundary of the dulness when the patient breathed deeply. Such oscillations are evidently mainly due to the action of the diaphragm. If we grasp the rubber membrane from below, in our bottle on page 41, and draw it downward, the water will descend and the bal-

loon will expand. If we then release our grasp, the balloon will contract, and the water and membrane will reascend. By repeating this maneuver we shall cause an alternate fall and rise in the line of demarcation between the balloon and the water.

So long as the diaphragm is still convexed upward, it is evident that its contraction must cause the line of demarcation between the effusion and the lung to sink, while its subsequent relaxation will allow the lung to restore the line to its former position, and Ferber describes exactly this condition of things in the dog's chest. By cutting away the external intercostal tissues of a dog, he was able to observe the combined movements of the lung and diaphragm, and of the water which he had previously injected. He says: "While the diaphragm was still active and convexed upward the water fell with inspiration and rose with expiration. With dogs of medium size these oscillations amounted to an inch, with moderately deep inspiration." He concludes, therefore, that an absolute immobility of the line of dulness would indicate peripheral adhesions, or a complete paralysis of the diaphragm and intercostals. I quite agree with Ferber in this conclusion, so far as it goes. I think, however, that the action of the intercostals would have but relatively slight effect upon the height of the fluid, since their excursions are very limited. On the other hand, the action of the external and so-called accessory muscles of respiration would exert considerable influence upon the height of the fluid, since they would expand the entire cavity of the thorax. When the sides of a vessel are drawn out, the level of the fluid within sinks. Hence, if the chest wall were lifted outward by the external muscles, the lung would naturally expand laterally, and it would also be obliged to expand downward, to compensate for the sinkage of the effusion, or otherwise

the diaphragm below would be drawn up higher; and in that case, of course, the upper level of the fluid would neither rise nor fall.

If the diaphragm is bagged down by an effusion, but is still able to contract, it is evident that its muscular action will *elevate* the fluid. The lung above will then contract, and *expiration* will be the result. In 1874 I tested this point by irritating the phrenic nerve with electricity, and I found that such irritation produced an act of expiration when the diaphragm was bagged down by an injection. My conclusions, therefore, as stated on page 61, must be correct, for I there say that the respiratory "movements cannot alter the physical principles which obtain between lung and injection. At best, they can only oblige a mutual readjustment of position."

Friction Sound. — At a very early stage of pleurisy one often hears a friction sound which is not constant, is very transient, and may not persist over twenty-four hours. Some observers say that by carefully watching a case one will never fail to detect this sound. Having disappeared, the friction sound remains absent until the beginning of the absorption or convalescent stage, when it reappears in a magnified form, and then often persists for days, and even weeks.

As to the cause of the friction sound, some assert that it is produced by the roughened pleuræ sliding over each other, while others say that the pleural layers are bathed with a viscid fluid, and hence stick together, until, by the efforts of respiration, they are torn asunder with a report. Be that as it may, however, we are more interested at present in the manner of interruption of the friction sound than in the manner of its production. Among all the books which I have consulted upon this point, I find but one theory advanced to explain the sudden cessation of the friction sound in the first stage, and its reap-

pearance in the last stage of pleurisy. As all opinions are substantially alike, it will be sufficient for me to quote one as the type for all, and I will choose that of Oppolzer.

Oppolzer says: "As soon as a few ounces are poured into the pleural cavity, the pleural layers are held apart, and friction ceases."

Thus we meet our old friend once more between the lung and chest wall, like the ghost in the closet, which explains all the family eccentricities.

Again, Oppolzer says that the friction sound does not reappear until, as a result of resorption, the pleura pulmonalis and the pleura costalis come again into mutual contact. Such is the prevalent theory, and it strikes me that it is as profoundly inconsistent as it well can be. Let us analyze it a little. Oppolzer says that a very few ounces are sufficient, at the commencement, to immediately separate the pleural layers and stop the friction sound; and yet, at the beginning of convalescence, when the friction sound reappears, the presence of a great many ounces is insufficient to prevent it. Why should a few ounces be able to accomplish in the one case what many ounces fail to accomplish in the other? If the lung was removed from the breast wall by a few ounces of effusion, what force has brought it back again while a larger amount of fluid is still present? The theory is absurd in itself; and more than that, I have proved that at the time the first friction sound is heard and disappears there is *no* separation of the costal and pulmonic pleural membranes. One might challenge me, however, by saying that the membranes are at least separated by the effusion which is trickling downward from all the points of exudation. I think, however, that no one will pretend that this thin sheet of descending fluid, supposing it to exist, can be so intact all over the lung,

and so thick, that the pleural layers can nowhere rub against each other.

Of course I do not deny that the intervention of effusion, when it does occur, will stop a friction sound. Thus a friction sound in the back would cease on lying down, if there were sufficient fluid in the chest to produce the condition of Model IV. But this is aside from the point in discussion.

A friction sound is heard in the first stage of pleurisy, and disappears before the effusion has attained any considerable size. It reappears again in a later stage, when the effusion is very large. How shall I explain these facts?

Three conditions are essential to the production of a friction sound between two surfaces. These conditions are: —

I. Roughness of the surfaces.

II. Apposition of the surfaces.

III. Movement of the surfaces against each other.

Eliminate either one of these conditions and the friction sound ceases. All writers hitherto have eliminated apposition of the surfaces of the pleura, and I have shown that this will not hold good for all cases.

Supposing we eliminate *motion*. Will not the result of such elimination harmonize most perfectly with clinical experience? We have just discussed the question of respiratory movement during pleurisy, and we have seen that in the first stage of that disease the patient voluntarily restrains respiratory movements to avoid pain. Later, the respiration ceases by reason of the impairment of muscular action, and hence the cessation of the friction sound.

The fact that slight respiratory efforts may still be made by the patient without a friction sound would be no counter-argument to me, because with any given degree of roughness of the pleural membranes a certain

degree of motion is always necessary to the production of an audible friction sound, and hence the reply to this supposed objection to my theory would be that motion existed, but was insufficient to the end desired. So much for the disappearance of the friction sound; when will it reappear? Naturally when the muscles have so far recovered from their paralysis that they are capable of again producing thoracic movements. Friction sound is often the first physical sign of beginning convalescence, and may appear before the level of the exudation has changed in any degree. It means, then, that the cure is proceeding from without inward, and that the muscles are the first to herald the glad tidings through the friction sounds. The friction sound, therefore, is wrongly considered a symptom of the abatement of the effusion; it is simply diagnostic of recovered function in the muscles. Its appearance may be synchronous with beginning absorption, or may occur, as I have said, with the fluid still at its maximum.

Vocal Resonance and Respiratory Murmur. — At the beginning, and during the early stages of an exudation, the respiratory murmur over the collapsing lung becomes fainter than normal, and in some cases it entirely vanishes. Sometimes one hears what the Germans call "indeterminate respiration," which is not sufficiently marked in character to be designated as bronchial. In such cases there are usually slight indications of broncophony, and perhaps of œgophony.

As the effusion becomes still larger, and the lung therewith becomes still more contracted, one hears both bronchial respiration and broncophony, and these sounds may be so intense that they are audible far down over the parts occupied by the fluid.

I think the explanation of these signs is as follows: Under normal conditions the combined vibrations of the

intra-pulmonic air, and of the lung and chest wall, produce the phenomenon of vesicular respiratory murmur.

The sound produced by a vibrating body depends upon the relative tension of the different constituents which compose that body. A membrane which is tense will transmit sound waves, which are imparted to it from the air, with comparative ease, while a relaxed membrane is unable to vibrate in unison with the air, and hence it dampens sound, and may prevent faint vibrations from being perceptible at all.

Any causes, therefore, which affect the relative tension of the lung and chest wall, must modify the facility with which those parts transmit the vibrations of the air within.

When an exudation, therefore, occurs into the pleural cavity, the lung retracts, and its relaxed tissue is no longer able to transmit the faint respiratory and vocal vibrations.

With the continued increase of the exudation, however, the lung retracts still more, the alveoli are contracted, and partially or wholly emptied of air, until a condition which is analogous to an inflammatory infiltration of the lung is established, and we then have bronchial respiration and broncophony, as in pneumonia.

Fraentzel says: "Ordinarily one hears indeterminate respiration when the lung tissue is not too strongly compressed by the effusion. As the compression becomes greater, bronchial respiration appears, provided the lung is not too far removed by fluid from the thoracic wall, and the bronchii are still pervious. Finally, the respiratory murmur may entirely disappear when the lung is too far removed from the chest wall, and when the bronchii are impervious."

I need hardly say that this explanation will not apply to the majority of cases, since the symptoms which we

are discussing are observed in the vertical position of the body, and at a time when the lung is not removed from the chest wall by an interposing layer of fluid.

We often notice that the augmented respiratory murmur and the broncophony, which accompany great contraction of the lung with large effusions, are audible far down alongside the fluid, and may even be heard at the bottom of the chest. Many authors think that this sound is transmitted through the effusion, and Bacelli bases a differential diagnosis regarding the nature of the fluid upon this theory. He says that sounds are transmitted best in the thinnest fluid, less distinctly in the thick, and not at all in the thickest.

I think that Bacelli's theory is wrong. A fluid does certainly transmit sound waves with greater intensity than air does, provided those waves are primarily generated within the fluid itself. If the sound waves are generated in the air, however, and are thence thrown upon the surface of a fluid, they do not produce equally intense movements within that fluid, and consequently the ordinary sounds of the external world are inaudible to a man under water.

When broncophony, etc., are heard very plainly, therefore, outside of an effusion and low down on the chest, I think it is because the sound waves are transmitted outward with such intensity by the lung that the whole chest well, and not merely a small section of it, is set in vibration, and hence the ear, even though applied low down, is still conscious of the vibrations within the chest.

The most recent theory concerning diminished respiratory murmur is that advanced by Dr. Carter in the "Birmingham Medical Review" for last July. He says; "When a lung is compressed by fluids and driven up against the spine, it is not without great displacement and disturbance of the bronchi. They are doubled up, and obviously they are first doubled up

where they are most slender, and when doubled up the vesicles at once lose their to and fro supply of air, and vesicular breathing for that part of the lung so affected is at an end." It is possible that this hypothetical condition of the bronchi might obtain, *if* the lung were compressed from below upward by an effusion. Considering, however, that compression is possible only with enormous effusions or in cases where the lung parenchyma is extensively involved by disease, and that enfeebled respiratory murmur characterizes early stages of pleurisy, I think the explanation will hardly suffice.

Certain other phenomena of pleurisy, incidental to the operation of thoracentesis, require explanation. Dr. F. I. Knight informs me that he has seen cases where a free discharge of exudation followed the simple puncture of the chest with a canula, and he asks how I explain this fact if the exudation is suspended by the lifting force of the lung as I claim. Of course the free escape of fluid, which Dr. Knight refers to, is precisely analogous to the spontaneous discharge which occasionally occurs with an automatic perforation in empyema. Therefore my explanation, which follows, will apply to both classes of cases. I have said that the lifting force of the lung is equivalent to the weight of a certain amount of effusion, and when this amount is exceeded by further exudation, the excess of the fluid is free to act directly according to its specific gravity, and bags down the diaphragm. Moreover, as the effusion increases in size the lung diminishes in volume, and therefore the lifting force of that organ becomes less and less in proportion as its expansion diminishes. It follows, therefore, that the excess of the fluid increases in a twofold manner the moment we pass the point of equilibrium between elasticity and effusion.

Now this excess of effusion, which is free to bag down the diaphragm, is also free to escape through a canula or

automatic perforation when the opportunity is allowed it. Of course the discharge must cease when the excess of effusion is exhausted, or nearly so; and Dr. Knight assures me that this conclusion of mine coincides with his own experience, since he has observed that a discharge which was free at first, gradually diminishes, and finally ceases, although there is every evidence of a considerable amount of fluid still retained in the chest. If the canula be of sufficient size, so that air can enter the chest and release the imprisoned fluid, we may have, even after the excess is exhausted, an alternate influx of air and efflux of fluid, as happens when we invert a bottle of water.

If I remove the flask, on page 32, from the beaker beneath, and open the valve C, a bubble of air rushes in and a portion of water escapes. This substitution of air for water continues for a few moments, if the flask be held perfectly still in the vertical position, and then it ceases, although a certain amount of water remains within. If I draw out the nozzle of the flask until the orifice at B is capillary in size, and then repeat the above experiment, no air will enter and no water will escape. If the orifice, on the other hand, be made much larger, all the water will escape, and nothing but air will remain below the balloon.

As the excess of effusion flows out, the diaphragm becomes relieved of its burden, and rises up to replace the escaping fluid. If aspiration is applied, the diaphragm will often rise up in front of the inner end of the canula, and by closing that orifice like a valve, it will check further discharge.

Dr. Henry I. Bowditch tells me that this peculiar action of the diaphragm has been "the source of great affliction" to him. It has, indeed, caused him so much trouble that he is often tempted to tap higher, where he will be out of reach of the diaphragm.

If my theory is right, it is very evident that the escape of the excess of effusion, although it allows the diaphragm to rise, cannot affect the expansion of the lung, since that organ supports as much weight after the perforation as before. If the aspirator be now applied, and more effusion be forcibly withdrawn, the lung, provided no adhesions are present, will begin to expand by virtue of its internal atmospheric pressure. If the case is one of long standing, and the lung has become accustomed to the condition of contraction, the patient often experiences great discomfort, as evidenced by coughing, fainting, etc. These symptoms are readily understood. While the lung is in a state of partial collapse, its various tissues gradually accommodate themselves to a diminished tension. The air passages and many of the alveoli are partially or entirely empty of air. Only a small amount of blood traverses the blood-vessels. Stagnation is everywhere manifest. Of a sudden the lung is forcibly expanded by the action of the aspirator, and a complete revolution is produced within that organ. The keen, bracing air sweeps once more the passages long unused to it. Torrents of blood rush through the distended vessels. Nerve fibrillæ long accustomed to inertia are roughly stretched and racked until the patient cries out in his suffering or sinks unconscious from nervous shock. Death may, and often does, result, if aspiration be carried beyond this point; but Dr. Bowditch assures me that he never has had any such accident in his own practice, because he always cautions the patient to inform him of the slightest sensation of constriction or uneasiness, and at that moment he ceases to operate. This point is of vital importance, and should be carefully remembered by all who attempt the operation of thoracentesis.

Dieulafoy, in a recent article in the "Gazette Hebdomadaire," 12 October, 1877, says that the essential point in the operation of thoracentesis is never to withdraw more than 1,000 or 1,200 grammes — about two pints — of fluid at one sitting. Larger amounts of effusion should always be withdrawn by repeated operations, and this rule should be more rigorous still in cases where the pleurisy is complicated by lesions of the lung parenchyma itself. He reports five cases of thoracentesis followed by rapid death. Each of these cases was complicated either by extensive adhesions in one or both pleural cavities, or by catarrhal and tuberculous affections of the lungs themselves. Moreover, in each fatal case large amounts of effusion — 5,500 grammes, 5 litres, 3 litres, and so on — were removed at one sitting.

I shall next consider the subject of the displacing force of an effusion; and as this subject is an exceedingly important one, I shall devote an entire chapter to it.

CHAPTER XII.

UPON DISPLACEMENTS WITH PLEURITIC EFFUSIONS.

Lung. — The lung is displaced upward in pleurisy by virtue of its contractile energy. We have seen that an effusion, up to a certain point, exerts a negative pressure upon the lung, and we obtain evidence of this in the shape of the letter S curve of flatness. I am, however, unable to state the relative amount of effusion which must be present before a direct upward pressure can occur. Considering all the physiological and pathological variations which are possible in different chests, I think that we can form no general estimate of amount which will be of universal application. I will lay down one rule, however, which is based upon pure physical principles, and which, I think, will apply to every case of pleurisy which is not complicated by adhesions.

An effusion can never compress a lung upward —

I. Until the retractility of that organ is exhausted, either by contraction or by disease; and —

II. Until the effusion has a fixed point of support below, from which to operate.

Compression of the lung, therefore, is impossible so long as the diaphragm is elevated into the thorax, unless, of course, that membrane is held up by adhesions or by intra-abdominal pressure from tumors, gas, etc. When, therefore, in extreme cases, the diaphragm is bagged down as far as possible, we may begin to suspect compression. Even then, however, we have no right to as-

sume such compression until certain other conditions have been taken into account. The gradual effect of the continued contraction of the lung is to straighten out the letter S curve. On the other hand, the immediate effect of compression would be to obliterate that curve. So long, therefore, as we are able to trace a well-marked letter S on the chest, we may be certain that the lung is well out of reach of compression. In all the cases which I have examined, and in those of Dr. Ellis, the curve persisted to a very high point, and hence my inference, stated in a previous chapter, that compression of the lung by a pleural effusion is a very rare pathological occurrence.

I wish to warn those who may repeat my experiments from falling into error regarding the amount of the contractility of a lung. If cocoa butter is injected into a thorax, several hours must elapse, for the cooling of the body and butter, before the chest can be opened. I usually leave the animal undisturbed during half the day and over night, as one experiment proved to me that nine hours was insufficient for complete solidification of the butter in a room of ordinary temperature. If one has a freezer at his disposal, he can, of course, economize in time. Now, on opening the chest after the body is cold, one will often find the lungs doughy and resistant, and he may infer that they are in a state of complete collapse. This is by no means the case, however. They are simply congealed, like the butter. Warm the lung, or, what is better, open the chest before the dog has lost his own heat, and the lung will exhibit its real degree of contractility.

Diaphragm and Intercostal Spaces. — We know that the effusion exerts a downward and a lateral pressure, by virtue of its weight; but we have discovered that this pressure cannot bag down the diaphragm, or obliterate the intercostal depressions until it exceeds the lifting force of the lung. Hence, if we find the diaphragm

and intercostals but little displaced with a large effusion, or if we find them displaced early with a small effusion, we can only conclude that the retractility of the lung is greater in the first case than in the second, or that adhesions are present between the diaphragm and the base of the lung. Weil very appropriately calls attention to the fact that the sagging of the diaphragm may also be prevented by strong intra-abdominal pressure from tumors, meteorismus, ascites, etc.

In order to illustrate the confusion which exists regarding the influence of a pleural exudation upon the diaphragm, I will make a short quotation from Fraentzel, who in turn copies from Wintrich.

Fraentzel says, when speaking of large effusions which displace neighboring organs: "In such cases the diaphragm supports the entire weight of the effusion, combined with the counter-pressure of the elastic organs which are displaced above. The degree of pressure is proportional to the area pressed upon, and consequently the right diaphragm yields sooner than the left. The diaphragm of men is stronger and more resistant than that of women."

Now this entire clause is one hopeless misconception of the true state of affairs in the chest, as I can readily show by analyzing its various assertions in succession.

I. The weight of the effusion undoubtedly acts downward upon the diaphragm in the vertical position of the body, but, properly speaking, it is not supported by that membrane, since both diaphragm and effusion are supported by a force external to themselves. Moreover, since the diaphragm is convexed upward, its tensile retractility acts in the opposite direction, *i. e.*, downward, and therefore the resistance of that membrane and the weight of the effusion both act in the same direction. As the effusion increases in amount, therefore, the dia-

phragm retracts until it becomes entirely relaxed, when its tension is exhausted, and then it offers only the feeble resistance of its weight, which still acts from above downward. Beyond this point the diaphragm begins to bag, and if the effusion still increases, the membrane may ultimately be placed in a new state of tension from its downward convexity. It is obvious that its tensile retractility will then tend directly upward, and therefore, at this point, but not till then, can the diaphragm be said to support the weight of the effusion. I leave out of consideration the functional action of the diaphragmatic muscles, since those agents always act in harmony with the tensile retractility of the membrane. They may magnify the results, but they cannot alter the principles already explained. It is necessary, however, to take into account the resistance of the abdominal walls and viscera. As the diaphragm descends those organs are packed closer together, and therefore they may offer so much resistance that the diaphragm cannot be bagged down to the extent described above. In such cases the fluid above would evidently derive support from the abdominal organs below, but not from the diaphragm, since that membrane would itself be supported in the same manner as the fluid.

II. The amount of hydrostatic pressure is *not* proportional to the area of the surface pressed upon, but it is proportional to the area multiplied by the height of the fluid. A given amount of water spread out in a thin layer over a large surface will exert no more pounds of pressure upon that surface than the same water confined in a small perpendicular tube will exert upon the membrane which closes the bottom of the tube. If we imagine, therefore, a bilateral pleurisy with an exudation of equal rapidity on both sides, then the total pressure upon the two diaphragms will be equal. What the right dia-

phragm gains by greater extent of surface it loses in an inferior height of column, and *vice versa* for the left diaphragm. If the right diaphragm bags down sooner than the left, it cannot be due to excess of area. It may be due to a difference in the lifting force of the two lungs. I said in a previous chapter, however, that I should expect the right diaphragm to be more depressed than the left, because the pressure of the fluid upon the former would be abetted by the negative pressure of the weight of the liver beneath. We see, therefore, that theory and fact here coincide.

The last clause of our quotation, however, is the most startling of all.

III. "The diaphragm of men is stronger, and therefore more resistant than that of women." I was obliged to read this sentence several times before I could understand it. I finally concluded that Fraentzel intended to express his theory for an implied condition which he has observed. He has probably observed that the diaphragm is found depressed oftener, and with relatively smaller effusions in women than in men, and he explains it by saying that the diaphragm is more resistant in the latter than in the former. I find that Weil copies the same assertion from Wintrich, and explains it by saying that the diaphragm of the woman opposes a smaller area to the pressure of the effusion. Further criticism upon these explanations is unnecessary after the demonstration which I have just given of the true relations existing between a diaphragm and an effusion.

If the diaphragm becomes depressed earlier and oftener in women than in men, it must be because the lifting force of the lung of women is feebler than that of men. I can see no other probable cause for it.

Spleen. — The spleen is pushed downward and somewhat forward with large left-sided effusions. Fraentzel

says that this is ordinarily the case unless the spleen is bound by adhesions. I have never observed such displacement of the spleen, but I should expect it from the principles already explained. The diaphragm is bagged down when the amount of the effusion exceeds the lifting force of the lung, and the point of greatest depression will correspond to the line of greatest depth in the fluid. In Figure 20, we see the rubber membrane most depressed at C, because the water is deeper in the line C D than in the line A B. Consequently the bagging of the diaphragm will appear first, and will be most marked in the lower posterior part of the chest, and the bag, thus formed, will hang down just behind the spleen. As the effusion continues to accumulate, the bag becomes longer and thicker, and must inevitably push the spleen downward and forward, as was said above.

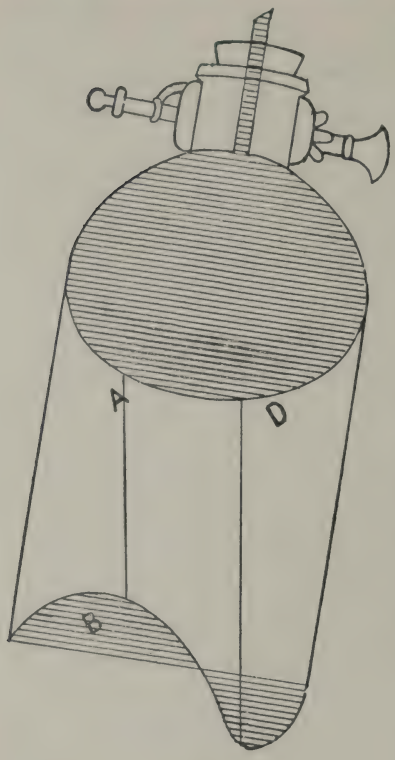


Fig. 20.

Liver. — The same reasoning may, I think, be applied to the downward depression of the liver, and to the tilting upon its long axis, which is sometimes observed

with right-sided effusions. As the excess of the effusion bags down behind the liver, it acts as a wedge, which produces the movements referred to. Ferber offers practically the same explanation for these displacements of the spleen and liver, and he gives a diagram representing the spleen tipped downward and forward by the sagging of the diaphragm behind it.

Stomach. — As the bagging of the diaphragm begins at its lower posterior attachments, and thence sinks gradually across the chest, the stomach is ultimately pushed downward, and the displacement of that organ is signalized by an obliteration of the tympanitic resonance in the so-called semi-lunar space of Traube.

The main portion of the stomach lies to the left of the median line, and well up against the diaphragm. When one percusses, therefore, over the lower part of the thorax in front, he obtains a tympanitic sound which proceeds from the stomach beneath. The area over which this resonance is obtained is bounded below by the inferior margin of the thorax, and above by a curved line with its concavity looking downward, and hence it is called by Traube the *semi-lunar space* of tympanitic resonance. It begins above with the fifth or sixth costal cartilage, and extends downward and outward to the anterior extremity of the ninth or tenth rib. Its greatest breadth amounts to four or four and one half inches.

The value of this semi-lunar space in the diagnosis of pleurisy has been variously estimated by different observers. Fraentzel considers it of the highest importance, especially in the differential diagnosis between pleurisy and pneumonia. On the other hand, Ferber thinks its testimony is very questionable, since one very often observes cases where quite large collections of effusion are present without any notable diminution of the semi-lunar space. Moreover, a slight diminution of this space may occur in cases where the lower lobe of the left

lung is enlarged by a pneumonic infiltration, especially if that lobe was previously emphysematous. Weil also suggests that this space may be diminished by filling the stomach and colon with solid or fluid substances.

I quite agree with Ferber and Weil in their doubts regarding the diagnostic value of this space. The depression of the diaphragm depends upon the excess of the effusion over the lifting force of the lung; hence, with a vigorous, unimpaired lung, we may have a large amount of effusion in the pleural cavity, and yet the resonance of the semi-lunar space may still be tympanitic. On the other hand, if the elasticity of the lung is seriously impaired by lesions of the pulmonic tissue itself, or by pneumonic solidification, a relatively small amount of effusion may completely obliterate the tympanitic resonance below. Ferber curtly dismisses the whole subject by saying that a pleurisy, which is large enough to diminish the semi-lunar space to any considerable degree, will make itself known by other phenomena of displacement. Weil is more kindly, however, and says that this space is worthy of some consideration, because in certain cases it gives us an idea of the increase or diminution of the effusion by the increase or diminution of its own size.

Ferber noticed a peculiar displacement of the stomach in two cases where he had produced an artificial hydrothorax of the left side. The fundus was pushed to the right, and the stomach was folded over on itself to a certain extent. A second and marked folding-in of the greater curvature occurred near the pylorus. Ferber says that this condition of the stomach, with left-sided pleural exudations, has been hitherto entirely neglected by authors, though it may sometimes be so great that complete flexion of that organ, with resulting stenosis, may occur. He thinks that the vomiting which is often observed, with excessive effusions, and which has been attributed to vio-

lent acts of coughing, may be due to this doubling over of the stomach.

HEART. — The displacement of the heart, with pleuritic effusions, is a very interesting and somewhat complex subject, which has occasioned much dispute among different authors. Guttmann says that large left-sided effusions, which compress the lung, push the heart over to the right of the sternum, and even as far as the mammillary line. Fraentzel says: "An effusion rarely reaches as high as the third rib on either side without a displacement of the heart in the opposite direction. Usually a smaller amount of fluid suffices for this." All of the authors whom I have consulted, with only one or two exceptions to be mentioned later, attribute this displacement to the direct pressure of the fluid. I think, however, that this explanation is entirely erroneous.

I have shown that an effusion does exert a downward and a lateral displacing pressure, but I have also proven that this pressure is incompetent to produce appreciable deviations of the diaphragm or of the intercostal tissues until there is a large excess of fluid over the lifting force of the lung. I have also demonstrated that this excess of fluid must act according to the simple principles of hydrostatics, and that it therefore produces the most palpable effects where it is deepest. A dam must be built very strong and very wide at its base, although its upper border may be as thin as a razor's edge.

It seems to me, therefore, very questionable to attribute so great displacing force to the upper layers of the fluid which lie alongside the heart. If a relatively large effusion is insufficient to push down the diaphragm, which is itself struggling to get down, how can the upper layers of that effusion push over the heavy heart? If the diaphragm were already depressed to its utmost, the effusion might, by further increase in amount, displace the heart,

or at least exaggerate any displacement which had already begun. In ordinary cases, however, the heart is always displaced long before the diaphragm has reached its point of lowest depression. We see, therefore, that the explanation given is wholly inadequate to explain the conditions observed.

The heart, with the pericardial sac, is suspended by the aorta, and this in turn is attached to the body of the third dorsal vertebra by ligamentous bands which, according to Luschka, are powerful enough to support many pounds weight without tearing. Below, the pericardial sac is attached, somewhat loosely, to the tendinous portion of the diaphragm by ligaments. This pendulous mass of tissues is placed between two highly elastic bodies which are striving to retract in opposite directions. The heart, therefore, being acted upon on either side by opposing forces, occupies a position where these forces just balance each other, and this is the status of physiological repose in the vertical position of the body. When the body assumes any inclination, the weight of the heart will immediately favor one or the other of the opposing forces, as the case may be, and hence that organ undergoes certain physiological displacements with every change of the body from the vertical position.

Now, when an effusion is poured into either chest, the lung of that side contracts, and thereby exhausts a certain amount of its retractile energy. The opposing lung, however, still remaining normal, immediately begins to draw the heart toward itself, and the degree of displacement thereby induced will be proportional to the diminution of energy in the compromised lung. The distance traversed by the heart during a displacement to the left with a right-sided effusion always is less than that observed on the right of the sternum with left-sided effusion, because the heart is already physiologically displaced to the left of the median line.

I have said that the migration of the heart is proportional to the diminution of energy in the contracted lung when the antagonistic lung is normal. If the lung of the opposite side, however, is itself deficient in retractile force by reason of structural lesions, the displacement will be much less marked. Guttmann says that the displacement is never so marked with pyo-pneumothorax as with ordinary effusions. The very existence of pyo-pneumothorax is usually evidence of parenchymatous changes in the lungs themselves, and I should therefore attribute the relatively slight movement of the heart to an enfeeblement of the elastic energy of the uncontracted lung. This point will be referred to again in the chapter on Pneumothorax.

I do not wish to be understood as saying that an effusion can *never* push against the heart. I merely mean to say that, practically, no pressure of the fluid upon the heart is possible at the stage when the displacement of that organ occurs. On the contrary, since the effusion must rise to replace the viscus as the latter is drawn over, it follows that the fluid must exert a negative pressure upon that organ, and thus limit the amount of displacement. Subsequently, if the effusion increases to a very great size, it may exert a direct pressure, and thus increase the displacement, but such cases are extremely rare.

As the lung of the unaffected side draws over the heart, its volume and its tension diminish, and we obtain evidence of this change by certain modifications of the percussion sound. Fraentzel says: "We have already explained . . . . that the sound lung is compressed laterally by the mediastinum which is forced over by the fluid when an effusion is large enough to depress the diaphragm, and this can be proved at any moment by percussion. The tension of the sound lung is thereby

diminished, and the percussion sound over it becomes abnormally deep and tympanitic."

We now know that it is impossible that the mediastinum, whether pushed or drawn over, should *compress* the lung of the well side, unless that organ were no longer able to contract. Had Fraentzel, therefore, substituted the word contraction and its correlatives for the word compression and its correlatives in the passage quoted, his theory would have been equally in harmony with facts and more consistent with the physical principles which we now know to prevail in the chest.

At this point I need hardly return to Fraentzel's theory of the counter-pressure from displaced organs above. Since those organs are not pushed over by the effusion, they cannot react upon the fluid or upon the diaphragm.

Not only the cause of the displacement of the heart, but also the mechanism of that process, has been the subject of much perplexity. Wintrich thinks that the heart swings like a pendulum from its base, and that its apex is therefore elevated with every deviation to the right or left. Skoda and Braune entertain similar ideas. Lebert says that the heart is first depressed by the sinking of the diaphragm, and then elevated by being pushed to the right. Gerhardt thinks that the heart is pushed over bodily with its apex always to the left of the base, with only rarely a rotation of its long axis. Guttmann says that the long axis of the heart preserves its normal inclination to the floor when that organ is displaced to the right. In only one instance did he see an exception to this, and then the heart was vertical in the right mammillary line.

Fraentzel also says, that in displacements to the right the heart is simply pushed over, and is never elevated, as Wintrich describes it. Ferber says, that the heart is first moved up bodily in the direction of its long axis and

against the aorta. Then the apex clears the diaphragm and swings to the right. This movement would necessarily produce a flexion in the large vessels, and Ferber observed such a twisting of the pulmonary artery in a case of artificial hydrothorax.

I have unfortunately neglected to note this point in connection with my injections, and therefore I can give no decided opinion. Ferber's theory, however, seems to me to be the most consistent with the anatomical structure of the parts, and with the fact that the heart is drawn, not pushed, over.

Size of an Effusion. — The question of the estimate of the size of an effusion presents itself naturally in connection with the displacement of neighboring organs. I have shown that the displacements in every direction depend upon, —

I. The retractile energy of the lung on the affected side.

II. Amount of the fluid present.

III. The retractile energy of the lung of the unaffected side.

A very large effusion, associated with a very powerful lung, will produce but slight displacements, while small effusions, when the lung of the affected side has lost its elasticity, will cause relatively great displacements. It is evident, therefore, that one can never form an accurate idea of the size of an effusion by simply observing the displacement of the heart or diaphragm. We have one sign, however, which is of the greatest value, and that is the letter S curve of flatness. Whenever we can trace this curve, we know absolutely the height of the effusion, and may then estimate its size by comparing that height with the position of the heart and diaphragm, and with the capacity of the chest. In forming our conclusions, however, we must furthermore take into account

the possible presence of adhesions. I have shown, however, that the persistence of a typical letter S curve is indicative of the probable absence of adhesions.

Replacements. — In closing this chapter I will briefly refer to the replacements which occur during the resolving stage of a pleuritic effusion. As the exudation begins to be absorbed, a potential vacuum is formed, which must be filled by other media. First and foremost, the diaphragm rises into the chest, and consequently if that membrane has been bagged down by an excess of effusion, its rise is the first indication of absorption. Then if the lung has escaped the entanglements of adhesions, it is again expanded by means of its internal atmospheric pressure. One might suppose that the internal atmospheric pressure of the well lung would also expand that organ and crowd over the mediastinum and its viscera, and this probably does occur to some extent. It must be remembered, however, that the lung of the affected side has lain in a contracted condition for days, and perhaps weeks, and consequently its power of resisting is weakened. Its supply of blood has been diminished and hence its structural integrity has suffered from loss of nutrition. Moreover, the very fact that the lung is contracted shows that its elasticity is diminished, and consequently it offers less resistance to the expanding air than the opposite lung, which would necessarily be placed in a state of great over-distention if it were crowded over to replace the fluid.

We always find, therefore, evidence of the expansion of the affected lung long before the heart and mediastinum are restored to place.

With the progress of the absorption the affected lung expands, until at last it occupies not only its normal position, but also a portion, at least, of the space abandoned by the heart. It is possible, also, that the diaphragm

will be more strongly arched upward, and thereby occupy some of that space. Be that as it may, however, as the lung of the affected side recovers its structural and functional integrity, it is obvious that the balance of power between the two lungs will swing in favor of that one which is now over-distended, and the heart will therefore be drawn back to its original position, where physiological equilibrium is again attained.

The case is very different, however, when the contracted lung is imprisoned by adhesions. In such a case the diminishing effusion is replaced, first, by the rise of the diaphragm ; secondly, by the heart and mediastinum, which are, of course, pushed over by the internal atmospheric pressure of the well side ; and thirdly, by the falling in of the chest walls from external atmospheric pressure.

CHAPTER XIII.

ABSORPTION OF PLEURAL EFFUSIONS.

Effusions have appeared in, and again disappeared from, men's chests during all time, and yet the mechanism of this pathological flow and ebb has been buried in the most profound obscurity. It may be that we know as little about this subject to-day as ever, but I wish to call attention to one theory relating to absorption which was propounded in 1866 by Dybkowsky. This theory is based upon an elaborate series of experiments, and bears upon its face the semblance of possibility at least.

At first Dybkowsky made a thorough study of the histological anatomy of the pleural membranes, in order to discover the course and distribution of the pleural lymphatics. He found that the portions of pleura which cover the diaphragm, mediastinum, and lung, are relatively poor in lymph vessels. On the other hand, the costal pleura is particularly rich in those vessels.

The pleural membrane consists chiefly of a single layer of epithelial cells, lying in immediate juxtaposition to each other, and of a sub-epithelial layer called the basement membrane. This basement membrane is a delicate network of connective tissue, and its interstices are occupied by capillary blood-vessels, and by the ultimate ramifications of the lymphatics. The open work among the meshes of the basement membrane, therefore, is called the lymph spaces. The lymph vessels do not open directly into these spaces, but form a closed tubular sys-

tem like the hæmatic capillaries. The epithelial cells, however, which form the walls of these minute vessels, are more or less spherical, and consequently, as they lie in contact with each other, they leave little intercellular openings, called stomata, which afford communication between the lymph spaces without and the interior of the lymph canals. The lymph spaces are separated from the pleural cavity only by the single layer of pleural epithelium ; and as the cells of this layer are very irregular in contour, they likewise present intercellular openings, which afford communication between the lymph spaces and the pleural cavity.

Having thus discovered direct channels of communication between the pleural cavity and lymph vessels, Dybkowsky proceeded to search for the forces which propel or attract an effusion along these channels.

He first cut out portions of the thoracic wall, and carefully injected the lymph spaces with a solution of Berlin blue. This fluid readily filtered through those spaces, but none of it entered the lymph vessels.

Then he injected a similar solution into the pleural cavity of a living dog, and after two or three hours he found the lymph vessels full of the injection, and the absorbing force was sufficient not only to draw fluid into the vessels, but also to take in solid particles of coloring matter. No absorption took place, however, if the animal was killed immediately after the injection. He inferred from these results that the muscular acts of respiration must have some influence upon absorption, and he explains his theory as follows : —

The lymphatics in the pleural membrane are situated between two forces acting in opposite directions, namely, the elastic lung on one side, and the intercostal muscles on the other. During expiration the intercostals bulge into the chest. During inspiration, however these mus-

cles contract and straighten, and thus exert a traction from within outwards upon the pleural membrane and its contained vessels. At the same time the elasticity of the lung exerts a traction in the opposite direction. This antagonism of these two forces tends to pull the different layers of the pleura apart, and as the walls of the lymph vessels are in close connection with the framework of the basement membrane, it also tends to separate those walls, and to form a vacuum within the same.

The moment this condition of affairs is established, the fluid within the pleural cavity, whether it be the result of the natural secretion of that cavity, or of a pathological exudation, rushes through the stomata, which I have described, and occupies the space formed. When expiration occurs again, the parts return to their former position, and the fluid absorbed is crowded along the lymph vessels to remoter parts, whence it is prevented from returning by the abundant valvular armament of the vessels. As indirect evidence of the correctness of his theory, Dybkowsky points to the fact that lymph vessels are found only in those parts of the costal pleura which cover the intercostal muscles, while the portions, which are reflected over the ribs, are destitute of such vessels.

Dybkowsky thinks that the process of osmosis may have something to do with absorption, but he believes that the chief work is accomplished by the antagonism between the intercostals and the lung during inspiration.

If this be true it is evidence that the rapidity and the amount of absorption must be proportional to the energy with which the intercostals contract, and Dybkowsky found that very little fluid, and *no* solid particles, were absorbed during quiet respiration. On the other hand, if means were taken to produce an abrupt, jerking inspiration, the absorption was proportionately great. Section of the vagus nerves will illustrate this point. Dyb-

kowsky also enclosed a dog's head in an air chamber and rarified the air. The intercostal spaces were exceedingly depressed, and each act of inspiration was accomplished only by a powerful initiatory jerk. The absorption was excessive.

Then he reversed the experiment, by allowing the animal to breathe into a chamber of compressed air. In this case the intercostal muscles bulged outward during expiration and retracted during inspiration. Of course, the conditions here were such that no antagonism between the intercostals and the lung was possible, and no absorption took place.

Dykowsky also found that fluids, which were injected into the intercostal tissues, always found their way into the pleural cavity. This phenomenon is analogous to that observed by Professor Ludwig, of Leipzig, who introduced a fluid, stained with Berlin blue, into the abdominal cavity, and then produced violent artificial acts of respiration. The lymphatics on the under side of the diaphragm became filled with the colored fluid. I have recently modified this experiment somewhat, by injecting a few ounces of colored fluid into the abdomen of a rabbit, and allowing the animal to breathe for himself. I also introduced a canula into the trachea, and, after a short time, I plugged the canula and allowed the animal to die of asphyxia. The acts of respiration were somewhat tumultuous during this stage of the experiment, and, on subsequent dissection, I found the lymphatics of the tendinous portion of the diaphragm beautifully conspicuous with their blue contents. It is evident, therefore, that the lungs and respiratory muscles must serve an excellent purpose in pumping secretions and exudations out of the abdominal cavity.

It is a noticeable fact, however, that, contrary to Dybkowsky's experience with injections into the intercostal

tissues, no fluid finds its way from the abdomen through the diaphragm into the pleural cavity. I am unable to state the reason for this exception. It may be due to some peculiarity in the structure of the lymph spaces or of the lymph vessels in the diaphragm, or it may be owing to the absence of stomata in the diaphragmatic pleura. The possibility of the absence of stomata is rendered somewhat probable by the fact that Dybkowsky never succeeded in obtaining, even with the most violent acts of respiration, any staining of the lymphatics of the diaphragm by fluid which had been injected into the pleural cavity.

Whatever may be the cause of this impermeability of the diaphragmatic pleura it certainly is a very fortunate provision, for, otherwise, every patient with ascites would rapidly pump his pleural cavities uncomfortably full of fluid.

Dybkowsky found that the lymph capillaries of the pleural membrane empty into larger lymph vessels which course along the grooves formed by the reflection of the fascia above and below each rib. Some of these vessels run back to the vertebral column where they unite with still larger ones. The majority of them, however, pass forward until they arrive at the sterno-costal muscles, then plunging into the connective tissue spaces of these muscles they continue along until they join the still larger vessels which accompany the mammary arteries. He concludes, therefore, that the lymph vessels which accompany the mammary arteries are the natural drainage ducts of the pleural cavity.

CHAPTER XIV.

PNEUMOTHORAX.

SUPPOSE a given volume of pleuritic exudation be suddenly converted into an equal volume of air. What will be the result? On page 36 I showed that the curvature of the lower part of the balloon was that represented by the curved line A B C when the balloon supported a column of air, and that the curve E B D represented the appearance of the balloon when supporting an equal column of water.

We saw that the conditions of antagonism which produced the curve A B C differed from those which produced the curve E B D, in that the weight of the column of air was equal to zero, as compared with the weight of the column of water and the lines l, l′, l″, etc., represent the effect of the negative pressure of the water. In every other respect, however, the principles involved in the two cases under consideration are identical. Supposing the supply of air to be increased indefinitely, the balloon will collapse, but compression of the balloon will be as impossible prior to the stage of complete collapse as it was shown to be in the case where we supposed an injection of water to be made, page 38.

The same must be true of the chest and its contents. If a perforation be made through the chest wall, or through the lung, so that air is admitted into the pleural cavity, the lung will collapse by virtue of its elasticity, but not by reason of compression. Subsequent to com-

plete collapse, compression may of course occur if more air be pumped into the chest by muscular action, and be there retained by any valvular peculiarity of the perforation.

In order to demonstrate these points more clearly, I repeated, with some modifications, the experiment of Damoiseau, which is described on page 72, and I obtained results altogether different from him. I killed a dog by puncturing his medulla, and then quickly but carefully removed all the external tissues from a number of the intercostal spaces. I thus exposed to view the costal pleura, and as this membrane is very thin, I could readily see the lung within. The trachea of the dog was unobstructed, and consequently the lung was in the position which it naturally assumed at the end of expiration. I then made a small opening through the pleural membrane, and admitted air into the pleural cavity. Immediately the lung began to retract, and this retraction was first manifested by a symmetrical elevation of the lower part of the lung. The air collected in one body between the lung and diaphragm, but no air, so far as I could see, penetrated between the lung and the lateral chest wall until the lower border of the lung had retracted upward to the distance of several ribs. Finally, as the lower part of the lung continued its ascent, its lateral surface began to recede from the chest wall, until a condition of complete collapse was established. By slowly reinflating the lung through the trachea, and then allowing it to retract again, I was enabled to repeat the experiment at will, and always with the same result. Moreover, as I had removed the external tissues from the sternum to the vertebral column in each intercostal space, I obtained a satisfactory view of the entire lower border of the lung, but I nowhere observed the appearance of a parobola, as described by Damoiseau. Before contraction of the lung

began, the inferior border of that organ was lower behind than in front, inasmuch as that is the natural shape of the chest cavity which the lung occupies. During contraction, the lower border of the lung receded from the costo-diaphragmatic groove throughout its entire extent. I noticed, however, that the posterior part of the lung contracted with greater rapidity than the anterior part; hence, as the lung rose, its lower border became gradually horizontal. We see, therefore, that the adjustments between the lung and the air were analogous to those obtained with fluid in the pleural cavity. The only variations observed were such as are readily explained by the absence of the negative pressure of a fluid in the one case, and its presence in the other.

In this connection I wish to call attention to a clinical fact which is significant. If one percusses the right side of a normal chest he will obtain a curved line of flatness corresponding to the transition from pulmonary resonance to hepatic flatness. On tracing this curve, one will find that it is very different from the Ellis curve, as portrayed on page 5. In the latter curve the letter S is sharply marked; its summit stands high in the axilla, and its anterior branch declines abruptly to the sternum. The hepatic line of flatness, on the other hand, is flattened out, so to speak. The letter S is visible, but is drawn out; the summit is low, and the anterior branch is about horizontal. Now, accepting the hepatic line of flatness as the boundary of the natural expansion of the lung, it follows that the modifications of that line, as shown in the Ellis curve, represent the effect of the negative pressure of the fluid effusion. The sharp decline of the Ellis curve toward the sternum would seem to indicate that the elastic energy of the anterior part of the lung is feeble as compared with that in the axillary re-

gion, and that therefore the former cannot appropriate so high a column of fluid to itself as the latter.

It seems now an almost axiomatic statement to say that the air is also powerless to exert any appreciable lateral displacing force until the lung is collapsed, and yet the heart is almost immediately displaced when an effusion of air occurs into the pleural cavity. The only possible cause for this early and constant displacement of the heart is the elastic force of the *opposing lung*, which draws those parts over to itself.

In order for the air in the pleural cavity to push the heart over, it would be necessary for that air to have some point of support, so to speak, from which it could push. A jack-screw would have very little effect in elevating a house, if its base rested on quicksands. How can the air brace itself against a fleeing lung?

I need hardly multiply arguments upon this point, and I will only add, that the air in the pleural cavity, though not the direct cause of the primary displacement of the heart, may nevertheless increase that displacement when it has accumulated in sufficient quantity. Moreover, I think the heart is more displaced in pneumothorax than in pleurisy, because the air, having practically no weight, cannot exert upon the heart that negative pressure which an effusion evidently would.

I wish to state here that this phase of the question of displacement of thoracic viscera in pleurisy and pneumothorax was original to myself, and was the direct outcome of my study of the flask and balloon. Subsequent reading revealed to me, however, that the same points had occurred to others before me, and I will therefore insert a few references to confirm the position which I have taken.

Skoda says, "The depression of the diaphragm is due *in part* to the weight of the fluid, but chiefly to the

diminished contractile energy of the retracted and diminished lung. The displacement of the mediastinum depends upon similar conditions. Since the traction of the lungs always affects both sides of the thorax, the moveable mediastinum must follow the lung which is still capable of contracting, and therefore, with right-sided exudations, the left lung will draw the parts over to itself. Only with excessive effusions in the pleural cavity (especially with pneumothorax) does the pressure of the fluid come into activity." So much for pleurisy. In connection with pneumothorax, he continues: "Air does not enter the pleural cavity simply at the cost of the torn and retracted lung, but the sound lung also retracts to such a degree as the mediastinum is movable, and it thus draws an additional portion of air into the increased space. The intense dyspnœa also contributes to this result. An excessive expansion of the chest, however, where the thoracic wall is bulged outward and the intercostal spaces protrude, does not occur until later, when the thoracic cavity has been stretched to its utmost by the contained air. . . . . An expansion of the air imprisoned in the pleural cavity by reason of increased temperature could probably produce only very slight effects."

In 1869, Dr. Powell, of England, published an article in the "Medical Times and Gazette" (January and February), in which he demonstrated a series of experiments illustrative of "the essential cause of the cardiac displacement in pneumothorax." His experiments are described as follows: —

1. "In the dead subject, the chest being healthy, a long and light needle was thrust vertically into the heart to serve as an index, and on the left pleura being then cautiously opened so as to freely admit air, the needle became slightly deflected, so as to indicate a movement

of the heart towards the right. An inspiration was now imitated by evenly raising both arms above the head, and the displacement became more marked."

2. "A similar experiment was made upon a living dog whilst under chloroform. The deflection of the needle was still more marked."

He then says: "The inference from these experiments was, that the elastic tension of one lung, when unopposed by that of the other, was sufficient to draw aside the mediastinum and with it the heart."

In a more recent article which I have received within a few days, and which is a reprint from volume lix. of the "Medico-Chirurgical Transactions," Dr. Powell continues with this subject, and gives an analysis of seventeen cases of pneumothorax in which he noticed the displacements and tested the pressure of the air within the chest.

In thirteen cases there was displacement of the heart, with different degrees of intra-pleural pressure. In three cases there was great displacement of the heart, but with *no* intra-pleural pressure. In one case there was no displacement of the heart, and this was accounted for "by the *unruptured* lung being so consolidated as not to permit of its collapse."

The conclusions which he draws from his observations and experiments are: —

1. "That displacement of the heart is an immediate and a most important sign of pneumothorax, depending upon the mere presence of air in the pleura and upon the contractility of the unruptured lung."

2. "That the cardiac displacement is by no means necessarily a sign of intra-pleural pressure, since the heart may be displaced to the right of the sternum, without there being any pressure."

3. "Hence, in discussing the question of paracentesis

in any given case of pneumothorax, we must take into consideration other things besides the position of the heart."

Dr. Powell's experiments have been criticised by Dr. Hayden, of Dublin, in his work on "Diseases of the Heart," page 100. As Dr. Powell's reply, however, seems eminently conclusive in regard to the incorrectness of Dr. Hayden's criticism, I will add one more quotation from the former. Dr. Powell says: "I must say that it is difficult to understand how there can be any difference between the atmospheric pressure within the lung and that outside the thorax; and the atmospheric pressure being the same, therefore, on both sides of the balance, but the elastic tension on the one side being annulled, the elastic tension on the other side seems to be the only force which, thus unopposed, can rightly be considered as disturbing the equilibrium. I would beg further to remark respecting two points in Dr. Hayden's experiments, undertaken in order to test the accuracy of my views, — firstly, that he apparently does not consider that the thoracic wall has anything to do with the question; and, secondly, he seems to think that by inflating the lung he can imitate its normal expansion. Thus Dr. Hayden, page 102, commences his experiment by removing the anterior wall of the chest of a subject, except the sternum, which he divides in a median line with a saw. He then detaches the root of the left lung, and corks up its main bronchus. He then further proceeds to inflate the right lung by blowing into the trachea to ascertain the effect upon the heart's position. Is it possible to conceive any experiment performed under conditions more diverse from those which are natural?"

Powell also saw that the mechanism of displacement of the heart in cases of fluid effusion into the pleura is

essentially the same as in pneumothorax, "but the occupation of the pleura by the effusion being gradual instead of sudden, as is its occupation by air, the displacement of the heart is slow instead of being instantaneous."

CHAPTER XV.

UPON THE HEART AND CIRCULATION.

PHYSIOLOGISTS have long been familiar with the fact that the retractile lungs exert a negative pressure upon the neighboring viscera and chest walls. The importance of this fact, however, has been overlooked by most writers, in their interpretation of the various phenomena of thoracic movements in health and disease. In 1863, Marey, of Paris, published a book upon the physiology of the circulation of the blood, and he demonstrated therein that the pulmonary retractility is a highly important factor in determining the movements of the heart and blood.

His experiments upon this point were conducted as follows: He introduced an elastic bag, which was attached to a long tube, into the various cavities of a horse's heart. The interior of the bag communicated through the tube with the interior of a rubber drum, which was attached to the other end of the tube. Every expansion or dilatation of the bag, therefore, caused corresponding vibrations in the membrane which closed the drum, and each movement of this membrane was recorded upon a revolving cylinder by a pen. He was thus able to obtain a graphic representation of every contraction and expansion of the heart itself, since the bag contracted and expanded simultaneously with that organ.

In this way he found that the pressure in the *right* au-

ricle is almost always *negative*, both in systole and diastole.

In the lower part of the right ventricle the pressure was never negative, but in the upper part of that cavity it was sometimes negative at the end of diastole. In the left ventricle the pressure was almost always sensibly negative during diastole.

Now the action of the cardiac muscles is mainly concentric. There are no fibres arranged so as to expand the heart by their contraction. Luton calls attention, however, to the fact that a hollow rubber ball which has been squeezed beyond the point of its natural contraction, will reëxpand a little by virtue of the elasticity of its walls, as soon as it is released from pressure. He thinks the walls of the ventricles may react in the same manner after the energetic systole. The amount of such expansion, however, must be very insignificant at best, and therefore, when the cavities of the heart are once contracted by the systolic movements, we must seek the forces which produce their diastolic expansion outside of that organ. An external force may operate upon the heart in one of two ways: —

I. It may be applied to the interior of the heart, and exert a direct pressure from within outward, or, —

II. It may be applied to the exterior of the heart, and act by traction or negative pressure.

When blood is driven into the heart from the venous system by a *vis a tergo*, the mechanism of the expansion of that organ comes under the first division of our category. No doubt the entrance of the blood into the heart is in a great measure due to the force of the venous circulation, and this force has been variously described and estimated by different physiologists. If this were the only force involved, however, the pressure within the cavities of the heart would *always* be positive, and

hence the pressure upon Marey's bag within those cavities would always have been positive.

On the other hand, if an external force were applied to the outer surface of the heart in such a manner as to draw its walls apart, the pressure within the cavities would become negative. Moreover, the influence of this negative pressure would extend into the venæ cavæ, and thus blood would be drawn into the heart by a *vis a fronte*, or by suction, as this process is familiarly designated. Marey found that the pressure in the right auricle is *always* negative in the horse, while the pressure in the other cavities is intermittingly so. He very naturally inferred, therefore, that the inert walls of the heart during diastole are drawn asunder by a negative pressure from without, and he seeks the force which produces this pressure in the retractile force of the antagonistic lungs. To quote his own words: "All the organs within the thorax, except the lungs, are subjected to a pressure which is less than that of the air. This potential vacuum is due to the retractility of the lungs, and is well demonstrated by the entrance of air into the pleural cavity when one makes an opening through the thoracic walls or diaphragm. . . . . The heart, being situated in this rarefied medium, is subjected to an aspiration which tends to dilate it when its contents are susceptible of a change of volume. . . . . The more the thoracic vacuum is increased, the stronger will be this aspiration. It will augment, therefore, each time that inspiration, by expanding the lungs, increases the elastic force of those organs."

The influence of the different acts of respiration is a very complicated question, and has been fully treated by Marey. I do not propose to discuss it at present, however, because it is somewhat aside from the immediate object of this chapter. I wish merely to draw attention

to the fact that the pulmonic retractility exerts a constant negative pressure upon the heart and its contents, and that the lungs must thereby, under physiological conditions, contribute to the rhythmical action of the heart, and to the emptying of the large cardiac veins.

Assuming the results of Marey's experiments to be correct, my friend, Dr. T. B. Curtis, of Boston, has suggested to me a very important deduction regarding the effect which a diminution of the pulmonary retractility would produce upon the heart and venous circulation. He thinks that "all diseases which enfeeble the aspiratory force of the lungs, by materially diminishing the retractile power of the pulmonary tissue, must necessarily be attended by disturbances of circulation, characterized by feeble arterial tension, together with venous repletion and stagnation." On searching various text-books upon this point, Dr. Curtis finds that such is actually the case, and he has kindly committed to me certain references and notes, which I will here append.

Lichtheim says: "As to the diminution of arterial pressure, observed in cases of pleuritic effusion, it is due to the direct pressure which the effusion exercises upon the heart, and it also depends upon the displacement of the large vascular trunks which empty into that organ."

Since I have shown that with ordinary effusions the fluid cannot exert a displacing pressure upon the heart, the diminution of arterial tension must be due simply to the diminished repletion of the heart during diastole. Less blood is drawn up into the heart, and hence there is less blood to be thrown into the arteries.

Fraentzel says: "Since a great part of the lesser circulation is rendered impervious by the compression exercised by the effusion, an impediment is thus presented to the outflow of the blood from the right ventricle and to its influx into the left ventricle; a venous engorgement

is thereby produced, which the right ventricle is incapable of overcoming by its ordinary efforts. Less blood reaches the arterial system, and consequently the pressure on the arterial walls is diminished, and coincidently there is a diminution in the quantity of urine secreted. If, however, the pleuritic effusion is suddenly let out, — as for example, by puncture, — the lung reëxpands quickly and the blood again circulates freely in the formerly compressed arteries, and thus, in a given time, a larger quantity of blood finds its way into the left ventricle, the arterial tension increases rapidly, while the pressure in the venous system as rapidly diminishes. As the pressure in the arterial system increases, so the quantity of urine increases, its specific gravity falls, the albumen disappears, etc."

Speaking of pneumothorax, the same author says: "Whenever pneumothorax is attended with much dyspnœa, more or less marked cyanosis frequently appears on the visible mucous membranes, as well as on the cheeks, nose, ears, and not seldom also on the hands and feet; . . . . simultaneously with the appearance of pneumothorax, and in pretty direct relation with the other symptoms of venous stasis, there is a diminution in the quantity of urine; it becomes red and dense, and often contains, as urine in venous congestion generally does, small quantities of albumen."

There are various points of interest in these two quotations. In the first place, the evidence which they contain of venous stagnation, as indicated by diminution of urine, presence of albumen in the urine, cyanosis, etc., are directly confirmatory of Dr. Curtis's *à priori* inference, and Lichtheim completes the picture by testifying to the diminished arterial pressure.

Fraentzel and Traube both attribute the venous stagnation to obstruction in the pulmonary circulation. The

pulmonary vessels are undoubtedly greatly reduced in calibre both in pleurisy and pneumothorax, when the lung becomes much contracted, and they must then offer a greater obstruction to the flow of blood than under normal conditions. This obstruction, however, can assume formidable proportions only when the heart sends more blood toward those vessels than they are able to accommodate. A small river bed is large enough for a small supply of water, though it may be severely taxed by a freshet.

Knowing, therefore, that the physiological antagonism in the action of the elastic lungs upon the heart does assist, to a great degree, in the diastolic repletion of that organ, we are justified in assuming that the symptoms enumerated are chiefly due to the disturbance in the antagonism of the lungs during pleurisy, rather than to the diminution in the calibre of the pulmonary vessels.

There is one other point of physiological interest which I will mention in connection with the heart. I have seen Professor Stricker, of Vienna, perform an experiment upon a dog, by which he showed that the blood tension in the aorta is greater than in the left ventricle. Professor Schiff, of Florence, told me that he had obtained similar results, but he doubted the accuracy of his records, because he employed a mercury manometer. He thinks that the mercury is too heavy, and therefore moves too slowly to execute a complete curve in the brief time required. Marey, with his more delicate "*cardiographe*," found that the maximum tension in the aorta is always slightly less than the maximum tension in the left ventricle. His curves show, however, that the mean tension of the blood in the aorta is much greater than the mean tension in the ventricle. I have made no experiments upon this point myself, and I mention it only to suggest that the lungs may be able to exert a more direct

and consequently greater influence upon the heart than upon the aorta, owing to the relative position of those organs. It seems to me, therefore, that the investigation of this point might throw some light upon the phenomena mentioned.

CHAPTER XVI.

SUMMARY.

In my argument regarding the relations existing between a lung, an effusion, and the thoracic walls, which consist of the ribs, the flexible intercostal tissues, and diaphragm, I have assumed that the internal and external atmospheric pressures are everywhere *potentially* equal to each other, and that, therefore, the adjustments between the effusion and the different organs mentioned are determined by the antagonism of the forces which are inherent in those organs, and especially by the balance of the retractile force in different parts of the lung.

I have thereby endeavored to prove that the immediate relations between a lung and an effusion are analogous to those which obtain between a column of water and the piston of a pump.

On the other hand, if we assume that the chest walls exclude totally the external atmospheric pressure, like iron walls, and if we ignore the retractility of the lung, then it is obvious that no effusion can enter the pleural cavity unless it be driven in by some supplementary force which is greater than the internal atmospheric pressure plus the weight of the lung. With such premises, however, we must also assume that the force of exudation in a case of pleurisy and the force of a simple transudation in a case of hydrothorax are each greater than the atmospheric pressure.

Such a condition of things, however, appears inconceivable to me. When we recognize that the blood which flows in the pleural membranes is subject to transmitted atmospheric pressure on all sides, and that the serum which escapes out of the vessels into the surrounding tissues is subject to a like pressure on all sides, and when we recognize further that this transmitted atmospheric pressure is less on the side toward the lung than it is on the opposite side of the pleural membrane, by the amount of the retractile force of the lung, it seems to me that the entrance of the effusion into the pleural cavity is precisely analogous to the entrance of cocoa butter into the dog's chest in the experiment described upon page 52, and that the lung with its retractile force is therefore comparable to the piston of a pump, which may be lifted by the feeble force of a child's arm, rather than to a piston of a hydraulic press, which is driven up by direct hydraulic pressure.

If I have not succeeded in proving my premises, however, then the criticisms, which I have made upon the theories hitherto prevailing, must revert upon myself.

If I have obscured my meaning by improper expressions or by language which conveys impressions not intended, that is my misfortune, but it is a misfortune from which few writers escape.

It will be noticed that I have refrained from stating definitely the amount which a lung can lift. I have purposely avoided this point, because I think that nothing is more fallacious, or more productive of confusion and error, than are numerical estimates which are based on a limited number of observations. In a problem which involves so many pathological and physiological deviations and complications, it would be presumption in me to lay down arbitrary figures until I have accumulated a very large series of observations as a basis for the same.

I have contented myself, therefore, with stating that a normal lung is capable of lifting a relatively large volume of fluid as shown by my models. Other physiologists have found that the contractility of the lungs is equivalent to about 6 mm. of mercury as indicated by a manometer attached to the trachea. I am preparing a series of experiments by which I hope to obtain quantitative estimates which will form the subject of a subsequent essay.

In conclusion, I will append a brief summary of the main points which I have striven to demonstrate in this book.

I have shown clinically and experimentally, —

(1.) That the letter S curve of flatness was first accurately described and traced through its various modifications by Prof. Calvin Ellis, of Boston.

(2.) That the letter S curve can be traced only in the erect position, and when the play of the lung is not hampered by adhesions; and that its persistence throughout the various stages of an effusion indicates the absence of adhesions in the lower part of the chest.

(3.) That the letter S curve of flatness corresponds in shape to the lower border of the lung, and in position to the line of apposition between the lower border of the lung and the upper border of effusion.

(4.) That the letter S curve is pathognomonic of a fluid effusion in the pleural cavity, but that it is impossible to judge from any variations in the curve as to the nature of the fluid present.

(5.) That the *dull triangle* which I have described corresponds to the posterior inferior part of the lung, and that this portion of the lung is not, in the erect position, separated from the chest wall by effusion until the amount of fluid has become relatively very large.

(6.) That a recognition of the dull triangle is very

important for the detection of the curve of flatness, especially in cases of hydrothorax, where the neglect of this region has led to the general but erroneous idea that the surface of a pleural transudation is horizontal.

(7.) That an effusion does not immediately intrude between the lung and the lateral chest wall, but that such intrusion occurs last of all, whatever be the position of the patient.

(8.) That a pleuritic exudation does not compress the lung in the manner universally taught, but that, on the contrary, the effusion exerts a *negative pressure* by virtue of its weight.

(9.) That the lower part of the lung does not become first compressed and then plunged into the fluid beneath, but that the entire lung contracts symmetrically throughout.

(10.) That the lung does not, properly speaking, swim upon an effusion, but that, by virtue of its retractility, it supports the entire body of the effusion, together with the diaphragm, until the weight of the fluid exceeds the lifting force of the lung.

(11.) That the position and shape which the lung assumes when associated with an effusion are determined by the balance between the weight of the fluid and the elasticity of the lung.

(12.) That the position and shape which the effusion assumes are determined by the varying degrees of retractility in different parts of the lung, and by the position of the patient, complications of course being left out of consideration.

(13.) That the excess of weight of an effusion is free to act upon the diaphragm according to its specific gravity.

(14.) That the diaphragm does not bag down until the weight of the effusion exceeds the lifting force of the lung, and the same holds good for obliteration of the intercostal depression.

(15.) That the heart, mediastinum, etc., are not pushed out of place by an effusion, whether of air or fluid, but that those parts are drawn over by the opposing lung. Enormous effusions may, of course, increase the displacement.

(16.) That friction sounds in the early stage of pleurisy are not interrupted by the effusion separating the lateral pleural surfaces, but that they cease because the respiratory muscles of the affected side are weakened and unable to cause sufficient motion for the production of those sounds.

(17.) That the negative pressure of the lung favors absorption *into* the pleural cavity.

(18.) That the action of the intercostal muscles favors absorption *out* of the pleural cavity during inspiration.

(19.) That the negative pressure of the lung favors the diastolic repletion of the heart, as shown by Marey and others, and that impairment of the retractility of the lung must therefore be accompanied by symptoms of imperfect heart supply, such as cardiac irregularity of action, diminished tension in arteries, and venous stagnation, as suggested by Dr. T. B. Curtis.

As I have previously stated, most of the points in this summary I consider to be original with myself, while others have been merely demonstrated in this book in an original and, as I think, conclusive manner. Many of the points appear to me now so axiomatic in their simplicity that I am amazed that they should have remained so long undiscovered, or, if they were known, that writers should hitherto have preserved so profound a silence in regard to them.

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ERRATA.

Page 18. Fig. 13, diagram 2. The sudden rise of the inner portion of the curve, near the sternum, shows the outline of the heart which lay below.

Page 57. Model IV. Second line. Instead of right intercostal space, read, right *ninth* intercostal space.

Page 74. Sixth line from top, instead of Experiment II., read Experiment I.

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